VOLUME 29 — 1959–1960

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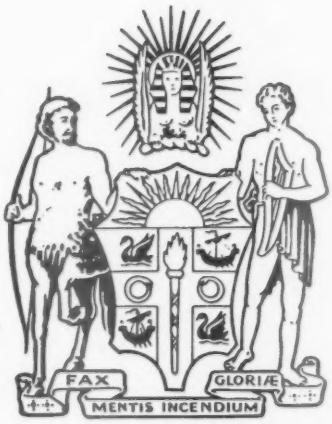

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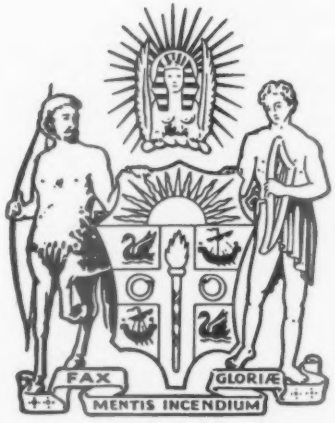

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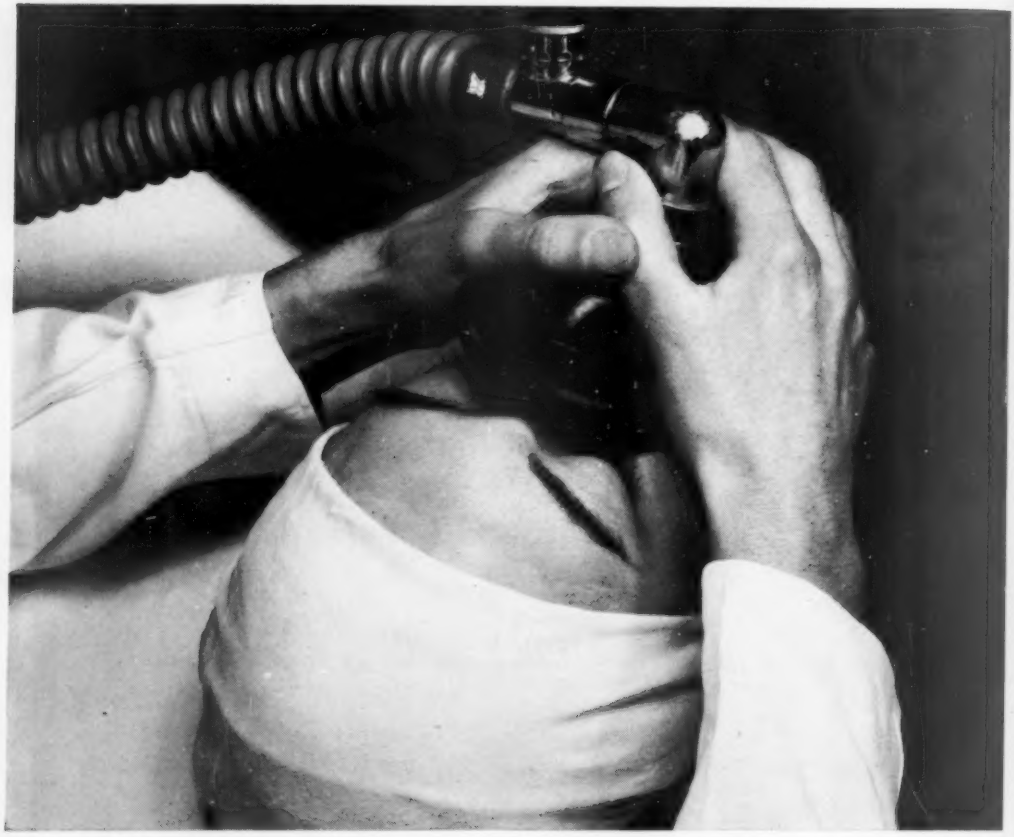
CONTENTS

August, 1959

		PAGE
An Appreciation	*Douglas Miller*	1
Memoirs of Sir Hugh Devine	*Raymond Hennessy*	4
Surgical Judgment	*Sir Hugh Devine*	8
The Roman Horace—His Health and His Poetry	*Sir Gordon Gordon-Taylor*	9
Pulsion Diverticula of the Esophagus and their Treatment	*O. Theron Clagett*	13
Surgical Considerations in Hiatus Hernia	*J. K. Clarebrough and J. L. Connell*	21
Experimentally Induced Tumours of the Intestine	*E. S. J. King and G. Varasdi*	38
The Best Technique in Gastrectomy for Ulcer	*V. J. Kinsella*	54
The Management of Overlooked and Recurrent Stones in the Bile Duct, with Special Reference to Transduodenal Choledochal Sphincterotomy	*Rodney Maingot*	66
Investigation and Preparation of the Jaundiced Patient prior to Operation	*I. J. Wood, S. Weiden, M. Ralston and I. R. Mackay*	77
The Pathology and Classification of Talipes	*Denis Browne*	85
The Role of Testicular Vascular Anatomy in the Salvage of High Undescended Testes	*R. Fowler, Jnr., and F. Douglas Stephens*	92
Rheumatoid Arthritis, Leg Ulceration and Positive Lupus Erythematosus Cell Test	*M. K. Benjamin and J. T. Hueston*	107
A Problem of Evolution	*Sir Gordon Bell*	111
Books Reviewed		114



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2. *Masterson, R. G., in discussion of Nix, F. G., et al.: J. Louisiana M. Soc., 108:395, 1956.*

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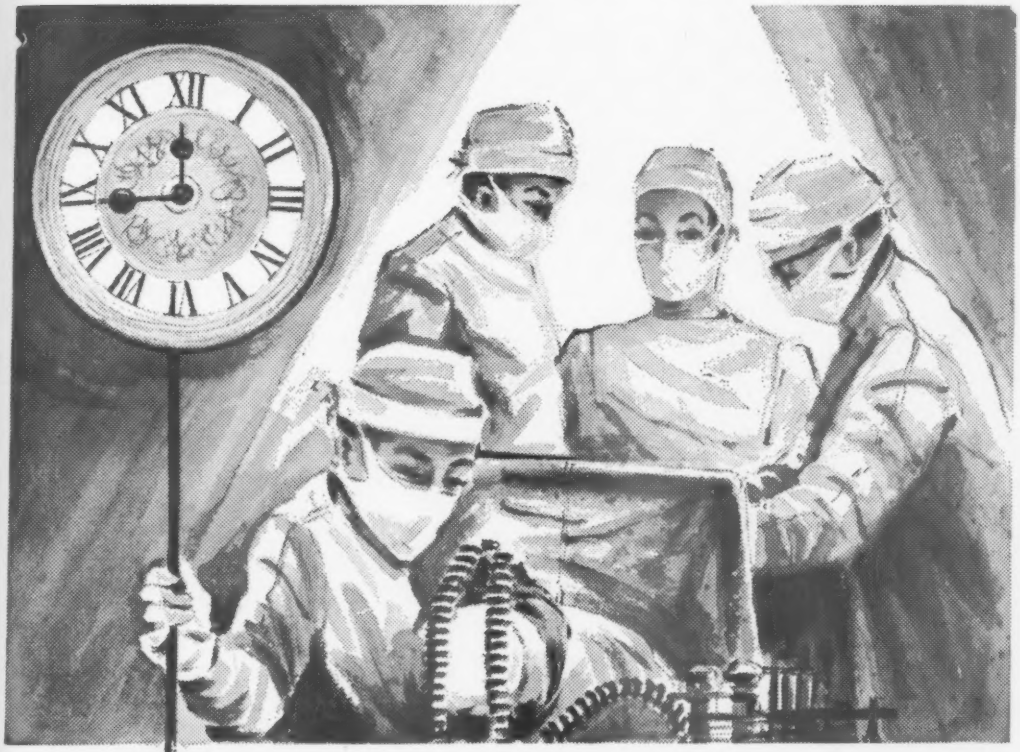

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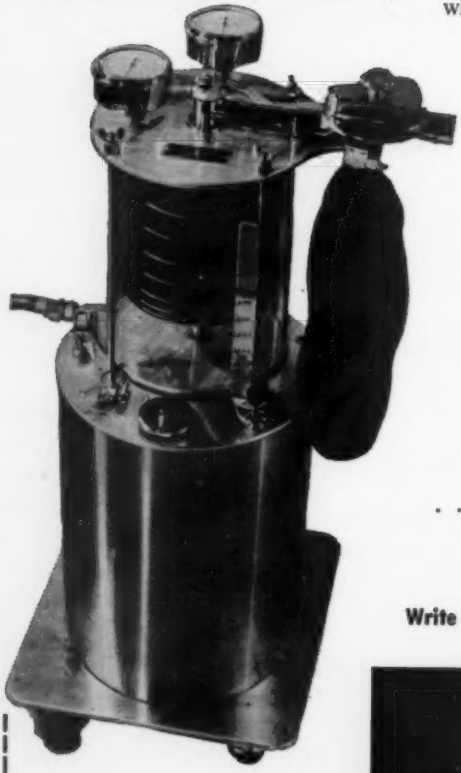

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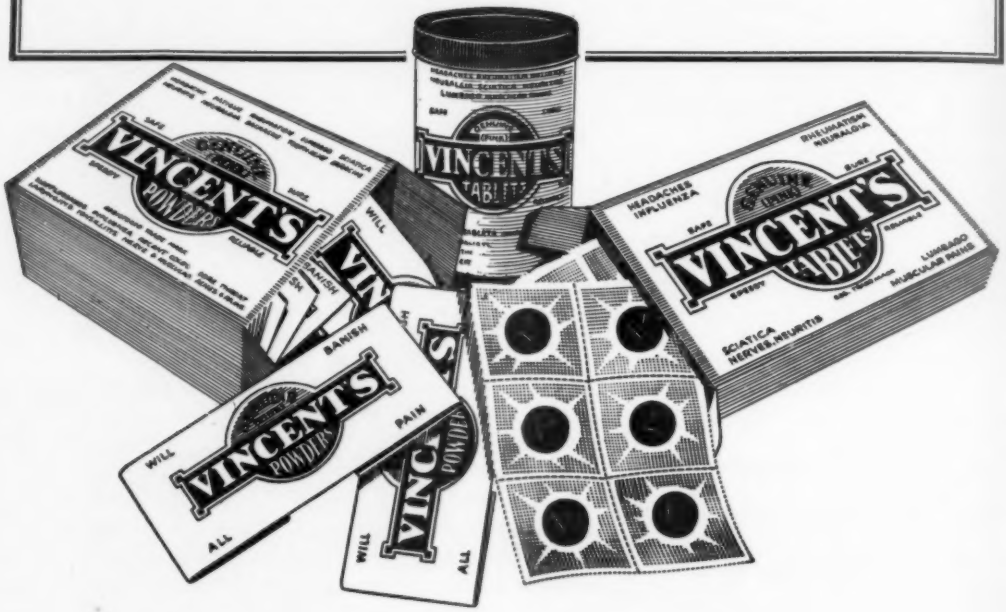

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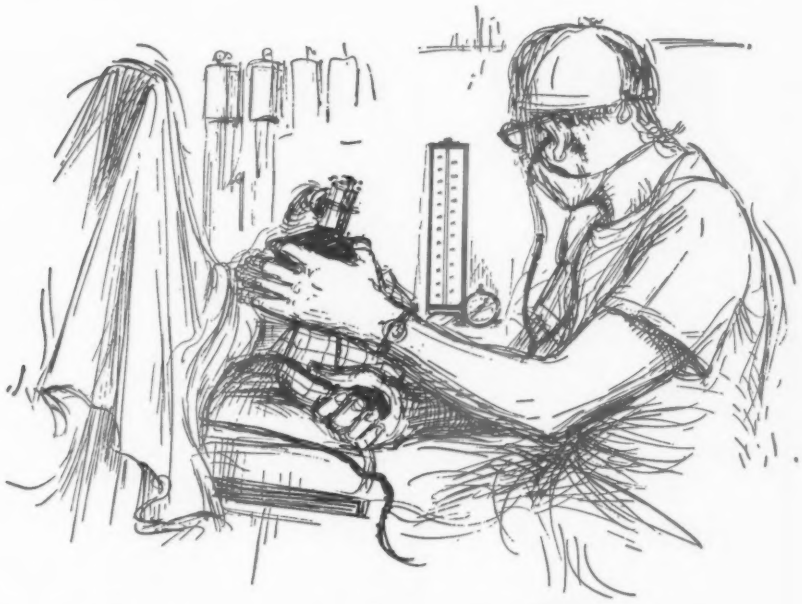

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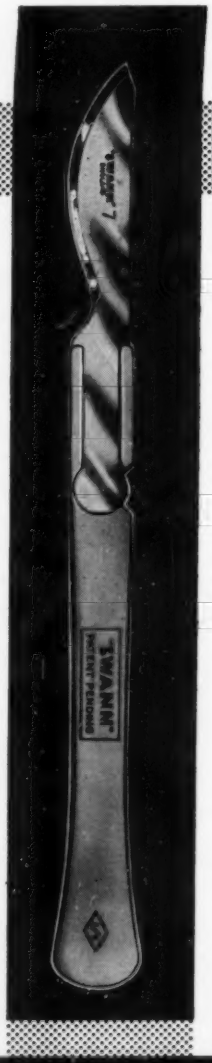

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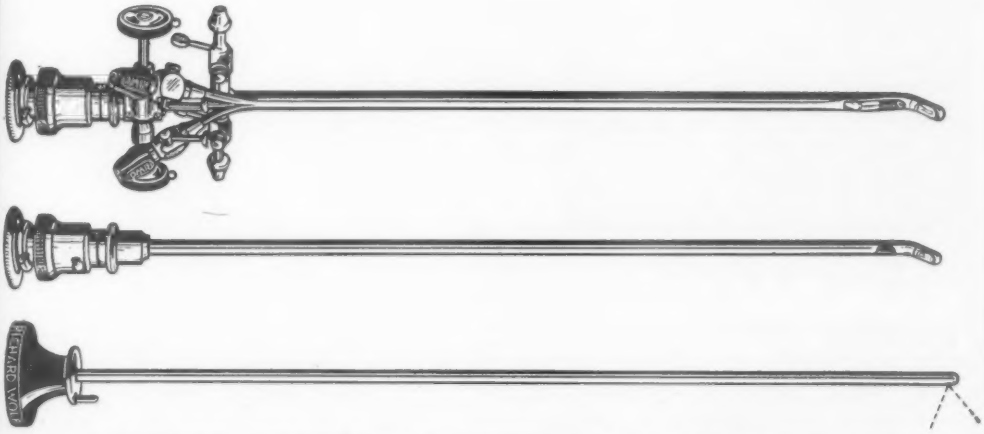

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Royal Australasian College of Surgeons

Sir Hugh Devine died on the 18th July, 1959

It is a sad coincidence that this announcement should be made in a number of the Journal published specially in his honour. Sir Hugh had greatly appreciated this gesture by the College, his friends and his admirers, and was looking forward keenly to its publication.

The interest of the many contributors at home and abroad had given him great pleasure and he often spoke of it during the weeks of his last illness.

May this number stand as a sincere tribute from the College to the memory of a great man of surgery.

Douglas Miller

HUGH BERCHMANS DEVINE

AN APPRECIATION

By DOUGLAS MILLER, F.R.C.S., F.R.A.C.S.

President, Royal Australasian College of Surgeons, 1959

THE portrait of Hugh Berchmans Devine reproduced in this Journal occupies by right a place of honour in the hall of the College. In it a skilful artist has captured a great deal of that unique personality.

In those keen eyes, that thoughtful brow, that aquiline face with its hint of great determination, can be sensed some of the attributes of one richly endowed with many natural gifts. The best of these was surely an enduring enthusiasm for the worthwhile things of his chosen way of life. This enthusiasm combined with a relentless drive and a restless search for better purposes and improved methods, has transcended all other influences in his life, and led to high achievement. So he has been able to make at the same time such a great success of the practice of surgery that his reputation is world wide, to perform a major role in the foundation of a great national institution and to found an important surgical journal.

His name is known and respected wherever surgery is practised or studied, his person is honoured and revered wherever he has worked or taught. In his time he has exercised a great influence particularly on the surgery of this country.

I write as one on whom his life has made a considerable impact. I first heard of him when I was a junior student in Sydney and his fame as an operator was a matter of common conversation in student circles. A few years later when assisting Sir Alexander MacCormick I met him when he had come to Sydney specially to see that senior master at work. Then I felt the stimulus of that avid student-like attitude and enthusiasm as he watched every point of technique and moved restlessly about the theatre making a critical appraisal of all he saw. I thought that keen face was young for one so famous and I liked his friendliness and sense of fun. I could feel then his power to lift people into the orbit of his own enthusiasm and this I often felt again at those surgical meetings where Devine was always a focal point of interest if there was something worth hearing or saying.

He had a striking faculty of being able to regard himself very objectively in both critical and admiring moods. He could look on his work and see that it was good. He did not however know complacency, or feel its stultifying influence.

His many books and papers were redolent of his original approach to his subject; his textbook *The Surgery of the Alimentary Tract* being a great contribution to surgical thought and teaching. It was acclaimed throughout the world because its concept was original, its source authentic personal experience and its presentation refreshing and novel. Here was no traditional documentary textbook but an account of the life work of a keen and original observer, an insatiable enquirer and an ingenious and skilled operator.

He travelled to many lands and was honoured and welcomed as one well known. Today still, in the United Kingdom, the United States, in Europe, India and furthest China I have found the recognition of Australian surgery linked with the name of Devine and that to have been his friend or student is to have a surgical passport.

All this was not enough for one who felt in his heart the great call to the responsibility which should accompany professional eminence. So it was that his discontent with things as he found them, his drive and genius, inspired in great measure the foundation of our College and his dedicated enthusiasm and faith in its destiny led to success.

With his distinguished colleagues Louis Barnett and George Syme, he was worried by the low standard of surgery being practised by the untrained and the inept and by the absence in our country of any authoritarian surgical body competent to teach and accredit to the

highest standards. They thought these things could be countered by the formation of an Australasian Surgical College. So it was that Sir George Syme, Hamilton Russell and Devine wrote an historic letter to some senior surgeons suggesting the formation of the College.

This College was to demand of its fellows a recognized standard of experience and training and high moral practice. It was destined to become the only body of men in Australasia accredited to practise major surgery. In a more distant vision it was seen to shoulder the responsibility of training and qualifying men to the highest level in our own country and under its own tutelage, an end which the dutiful efforts of fellows could bring within easy reach today.

In the early days the headquarters were planned for Canberra, but this was soon recognized as impracticable. It was then that Devine's presentation of the case to Mr. Hogan, the Victorian Premier, obtained for the College a long lease of the present wonderful site which had been dedicated for purposes of education. The succeeding Government confirmed this action. Thus the unique site of our gracious building is a tribute both to Devine as an advocate and to the vision of these statesmen who recognized the national importance of the new college.

In many things Devine was fortunate but in nothing so much as in his close association with Alan Newton in these years of College formation. They were complimentary characters, Devine the tactitian, the negotiator, the friendly envoy; Newton, the aloof ruler, the elder statesman; the one brimful of ideas often struggling for articulation, the other articulate to the last syllable; the one with a whimsical gaiety, the other authoritarian, impressive.

They both enjoyed one another and their combined efforts were irresistible in the attainment of their ideals. Devine has often told me of arguments and differences behind the scenes, "but we always presented a united front at meetings." He looks on this association as one of the greatest influences in the formation of our College.

In the early days of the College there was some doubt as to whether its spiritual affiliation was to be with the Royal College of Surgeons of England, or the American College. Natural centripetal forces became evident however with the significant gift by the Royal College of Surgeons of our great mace and in the fruits of a trip to London by Devine from which he returned with the coat of arms, the prefix Royal and an ambition to acquire a Royal charter. Later our common sympathies and aims were signalized by the visit of Sir Holburt Waring, when he was President, to open our College building, in the reciprocity of our primary examinations and the concessions we have made to Fellows of the senior College. The conferment of an honorary fellowship of the English College on Devine was a tribute to his influence in this direction.

From these early days Devine worked unceasingly for the College, he served a term as president and eventually retired from the Council in 1948. His retirement was prompted by a wish to make way for younger men but at any age it would have been difficult to replace him.

I cherish the memory of those last days when I had the privilege of sitting on the Council with Divine, Newton and Hailes and feeling their dedicated spirits. In Devine's letter of retirement to the President he wrote, "You will, I am sure, understand that after being a member of the Council of the College from the first moment of its existence, I cannot help feeling a little sorrowful when I ask you to accept my resignation as a Councillor. I feel however that I must make way for those who, because of their station in the world of surgery, should come to the Council with fresh minds, new ideals and rich in energy. I shall leave the Council with the confidence that its professional principles are so right that it will always attract the best surgical minds, and with such guidance the College will always be the highest influence for good in the profession of surgery."

The fulfilment of that prophesy will be Devine's great legacy to the surgery and the welfare of the people of this country.

At the first meeting of the College in Canberra, It became apparent to him that the College needed a mouthpiece, an organ devoted to the betterment and information of surgeons and he conceived the idea of founding this Journal. He collected much of the material which

had been supplied at the first scientific meeting and soon had enough to fill two numbers. He then collected advertisements and money to pay for it. Having done all this he submitted the papers to the President, Sir George Syme, to inspect and got his formal permission to run the journal for eighteen months. It quickly flourished and remained under his Chairmanship until 1949. It was rather characteristic of Devine that he organized and founded the journal without waiting on formal permission from the Council and it is of interest that the minutes of the College contain no record that this was ever given. No task was too great or too small in the service of the journal, from the writing of a paper to the soliciting of an advertisement. "It was a joy to me," he wrote.

His hospital life was centred on St. Vincent's where he shared the work and developed the fame of that hospital with such worthy contemporaries as Dunhill, Julian Smith and Murray Morton. Many a student and graduate of St. Vincent's must have kindled his first flame of professional excitement at the fire of Devine.

I could not write of Devine's professional life without mention of the brilliant and versatile son who meant so much to him and on whom he built great hopes, fulfilled ahead of time, when John, very young but almost as an equal, joined him in his practice, his writing and his thinking. Such phenomenal fulfilment of hopes was too soon darkened by his tragic death. Devine's philosophy and mental timbre strengthened him to bear even this and his interest in the great things of surgery and of science, shone as strongly as ever.

Now in the evening of life he is hale and active, still bubbling over with ideas, never knowing the weariness or fret of age, not puffed up by his great past, but ever thinking for the future and the good of this College and this Journal which were so very much his own creations.

His Royal Highness the Duke of Gloucester presenting the Diploma of Fellowship of the Royal College of Surgeons of England to Sir Hugh Devine at Government House, Melbourne, on 23rd of February, 1945.

MEMOIRS OF SIR HUGH DEVINE

By Raymond Hennessy
D.D.Sc., F.R.C.S. (Edin.), F.R.C.S., F.R.A.C.S.
Melbourne

ONE of our national poets has observed that the child is father to the man. This is admirably illustrated in the subject of this memoir, Hugh Berchmans Devine, in whom was inborn an indefatigable pursuit of knowledge, relentless ambition and an inflexible determination to excel. He was born in 1878. His father John was a grazier in the Werribee district where he was reared. After education at St. Patrick's College, Ballarat, he matriculated but unfortunately without Latin, at that time a compulsory subject for the medical course.

After a country apprenticeship in pharmacy he became employed by Henry Francis, a prominent and old established pharmacist in Bourke Street, Melbourne. This pharmacy was a kind of meeting place for doctors and personalities of other professions; by their conversation and contact he was able to develop his interest in medicine which was his life's ambition. He recalls that a frequent visitor was Sir Thomas Fitzgerald, perhaps the most famous surgeon at this time, who was wont to include a visit to the pharmacy in his early morning walk.

Having completed his studies in pharmacy and no doubt with a mind already broadened and inquisitive, Hugh Devine began his medical studies at the University of Melbourne, and after a brilliant course graduated in 1906, winning the Beaney Prize in Surgery. His position in the final honours list enabled him, after serving the usual twelve months term as resident medical officer at the Melbourne Hospital, to become acting superintendent of this hospital.

In conjunction with Dr. W. Silberberg he established a register system for the classification of diseases from hospital records. This was the first attempt to use the clinical material of the hospital on an organized basis. The system they devised proved so satisfactory that it remained in use for about twenty years.

About this time he became associated with the late Sir Thomas Dunhill, whose brother was Devine's study mate at Queen's College, and who was then beginning his career at St. Vincent's Hospital. Dunhill introduced Devine to the Mother Superior and persuaded him to join the staff of the young hospital; soon afterwards to be enlarged to one hundred beds and become a clinical teaching school attached to the University.

Here in St. Vincent's Hospital he worked and shared in the development of this renowned clinical school. He was indeed an inspiration to many a student and graduate who had the good fortune to be associated with him there.

At this time he was eagerly working at anything appertaining to his projected surgical career and was much influenced by two men, Sir Harry Allen who was an enthusiastic and proficient dissector in the autopsy room, and Professor Watson of Adelaide, an original thinker in applied anatomy and pathology and a stimulating and provocative teacher. This extraordinary man Watson seems to have influenced everybody who came in contact with him.

Devine's next move was to go abroad in 1911. He made Vienna his headquarters and established himself at the *Allgemeines Krankenhaus*, a famous hospital founded in 1765. Here he found the method of teaching pathology direct from fresh autopsy specimens and the operative surgical classes on recent cadavers, a complete realization of his own ideas of acquiring knowledge. As this hospital had over 2,000 beds the material for study was inexhaustible. One of the features of the Viennese Medical School at this time was the study of clinical roentgenology, then in a very early stage of its development. The diagnosis of gastro-intestinal disease by X-ray meals of bismuth and subsequently barium was being closely investigated. Holzknecht, then world famous, was in charge of this

department. This fascinated Devine who was quick to realize the marvellous possibilities of X-ray for accurate pre-operative diagnosis of gastric diseases, and subsequently by long experience, he became expert in this work. He had already decided that gastric surgery was to be his special study and he was determined to play a part in its development.

After completing his studies in the United Kingdom and America, Devine returned to Melbourne and set up practice as a surgeon.

He obtained the degree of Master of Surgery of the University of Melbourne in 1913.

During the following seven years he developed his technique and experience in gastro-intestinal surgery, particularly as to assessing the significance of symptoms and the likelihood of their being relieved by surgical treatment. The account of his work during this period was described in a paper read in Brisbane in 1921. This paper has become one of the historic landmarks in the development of gastro-intestinal surgery in Australia.

At this British Medical Association Congress in Brisbane in 1921, Louis Barnett, a New Zealand surgeon, made the suggestion by letter that there should be a College of Surgeons in Australia on lines similar to the American College of Surgeons founded in 1912 which had done so much to improve the standard of surgery and organized surgical teaching in hospitals throughout the United States of America.

In 1924 William Mayo with Dr. Franklin Martin and three other American surgeons came to Australia on a goodwill visit and spent a morning at St. Vincent's Hospital watching Devine operate. The case was a difficult gastrectomy for extensive infiltrating non-maligant ulcer; this operation was done to the admiration and applause of the critical and distinguished onlookers. William Mayo described Devine's technique as distinctive in type, largely orignal in principle and method and almost perfect in execution. As a result of this, in the following year, Devine was invited to read a paper before the American College of Surgeons. The title of the paper was "Basic Principles and Supreme Difficulties of Gastric Surgery."

Returning to Australia Devine devoted his energies to the formation of the College of Surgeons. His contribution to this ideal is the subject of the remarks of the President of the Royal Australasian College of Surgeons in this, his number, of the Journal which he himself founded.

Early in his career Devine began to contribute to surgical literature culminating in his well-known textbook *The Surgery of the Alimentary Tract* which was published in 1940 with nearly 700 illustrations. It was most extensively and favourably reviewed in British and foreign literature. It was acclaimed by some reviewers as the greatest textbook on the technique of abdominal surgery since Moynihan. In conjunction with his son, the late John Devine, he also published another book entitled *The Surgery of the Colon and Rectum*; this too was favourably reviewed.

In the surgery of the large bowel Devine developed revolutionary methods in dealing with malignant disease of the right side of the colon. His first paper describing this was published in 1925 in Australia; a subsequent paper appearing in the *Lancet* in 1931.

In 1935 he published his now well-known method of synchronous removal of the rectum by two surgeons, one working from the abdomen and the other from the perineum. In 1937 he contributed a further paper on this subject to the *British Journal of Surgery*.

All the papers illustrate an energetic and original approach to the problems of abdominal surgery.

In 1927 Hugh Devine became obsessed with the idea of founding a journal along the lines of the *British Journal of Surgery* and *Surgery, Gynecology and Obstetrics*. He was entirely responsible for the contents of the first two numbers, not only editorially, but financially. The wisdom of establishing this journal, the *Australian and New Zealand of Surgery*, was not, at that time, apparent to some of his colleagues. Devine remained Chairman of the Editorial Committee for twenty years.

In 1936 he was knighted, and in 1945 he obtained the honorary fellowship of the Royal College of Surgeons of England at the hands

of the Duke of Gloucester, then Governor-General of Australia, who made a very laudatory and graceful speech about the recipient. While the Duke remained in Australia, Sir Hugh Devine was one of his honorary surgeons.

CHARACTERISTICS

Between 1920 and 1930 Devine reached the acme of his surgical skill and prowess. His operative technique had many original and individual features and was delightful to watch. He had by this time perfected his operating frame and retractor after many years of experimenting with various combinations and arrangements. The advantages and the necessity for this retractor are not so apparent to the present generation of surgeons who have become accustomed to operating on paralysed patients with flaccid abdominal muscles. The primitive anaesthetic methods Devine and his contemporaries had at their disposal made it very difficult to get adequate exposure in abdominal surgery without some mechanical retraction.

Another invention he introduced was a sucker. The present writer can recall him using one as early as 1917, the operating theatre tap being used as the suction apparatus. He was also responsible for use of large gauze wraps which he called scarves and which he used to entangle the intestines and push them away from the site of the operation. Another individual feature of his operative technique was the use of scissors rather than a scalpel for dissecting. He had by this time designed his spade pointed scissors with which he became most proficient in stripping tissue planes. The use for the scissors was seen at is best in difficult upper abdominal surgery with many adhesions. He also invented a portable kidney rest which fitted on the primitive operating tables of the period.

As an operator he was quick but not hurried, dexterous, confident and safe. His operations were well staged but if the exigencies of the situation required any sudden change of plans or if any unexpected emergency arose, his instinct to do the right thing was sure. He was fastidious about haemostasis. His suturing was accurate and tidy. His style was devoid of fuss or mannerisms and except under extreme provocation, he was never irascible nor impatient. He was easy to assist. It was always a matter for comment how seldom his patients developed post-operative complications even after the most difficult and prolonged operations. His repertoire of operations was versatile and included all routine operations of the abdomen, neck and limbs. He was supreme in the surgery of the thyroid gland and after Dunhill's permanent residence in London in 1921 his opportunities in this branch of surgery were unsurpassed in Melbourne.

His perspicacity in small things was remarkable and is illustrated by the mask he designed in 1916 and which he was accustomed to wear. It was made of several thicknesses of gauze stretched over a wire frame which stood some inches clear of the lips, tongue and nose, being attached to the ears by spectacle loops; it was readily sterilized by boiling. He realized even then that any mask touching the lips, tongue or nose was more dangerous than no mask.

His private clinic in Collins Street was complete in itself and included an operating surgery, comprehensive X-ray apparatus, and the facilities for the examination of pathological specimens. He elaborated a method of working with printed history sheets and blank spaces which made for the utmost efficiency without necessary loss of time; otherwise, despite a not inconsiderable staff, he could never have handled the enormous volume of work which at this time comprised his private practice.

His methods of teaching were characteristic; he would have a portable blackboard upon which he would illustrate his clinical talks by headings and drawings. His enthusiasm for teaching was probably superior to his natural gift for teaching, but by assiduous practice he eventually became a very clear and lucid exponent of the art.

He was punctual and exact about his appointments with his colleagues; always accessible and easy of approach.

Sir Hugh enjoyed good health and maintained his physique, naturally robust, by a regimen of abstemious habits and vigorous

week-end exercise. For many years he was accustomed to stop practising before Christmas and spend the next six weeks at his house at Flinders, charmingly situated on the shores of Western Port. Photography, golf, tennis, fishing and yachting were his hobbies. It was there too that, in his "leisure" hours, the manuscripts for his books were assembled and corrected.

Living now in Melbourne, he finds happiness with his family and his grandchildren. His alert mind is everactive with an up-to-date camera, new books, the latest developments in surgery and, above all, the welfare of the College.

The following honours have been bestowed on Sir Hugh Devine during his active surgical career: Honorary Fellowship of the Royal College of Surgeons of England; Honorary Fellowship of the Association of Surgeons of Great Britain and Ireland; Honorary Fellowship of the Royal Society of Medicine and its Proctological Section; Fellowship of the American College of Surgeons; Honorary Fellowship of the International College of Surgeons; Honorary Fellowship of the Greek Surgical Society; President of the Royal Australasian College of Surgeons, and Chairman of the Editorial Committee of the *Australian and New Zealand Journal of Surgery.*

SURGICAL JUDGEMENT*

By Sir Hugh Devine

IN a surgeon there is no quality of mind more to be desired than that of judgement. Judgement is the product of a mind cultured by a liberal and a professional education and matured by experience. It is judgement even more than skill that makes a truly successful surgeon.

Judgement is not readily developed during under-graduate training when the academic foundations of a surgeon are being laid. In the comparatively short period of under-graduate training, the student is required to absorb a mass of accepted knowledge, presented to him with a dogmatism characteristic of under-graduate teaching. There is too little time for explanations which would enable him to assimilate it on a basis of physiology and pathology. The intensive teaching dulls any imagination he may have; and the result is that if he is to be academically successful he must form memorizing rather than reasoning habits of mind. Thus, though he may acquire much knowledge, he develops little wisdom and lays no foundation in the art of acquiring wisdom.

As time goes on, however, the developing surgeon begins to exchange his memorizing for reasoning habits of mind. In this way he begins to accumulate that mature wisdom and develop that sound judgment which is so essential in the make-up of a surgeon. He is encouraged in this as he comes under the influence of those who have attained a position in the profession which entitles them to hand on the torch of knowledge and to point the way of wisdom and judgement. The young surgeon becomes sensible of this influence first in the hospital wards, operating theatres, and laboratories, then at surgical conferences, and finally through the medium of surgical literature, in relation to which he must cultivate a kindly critical mind.

Chastened by his experiences, enlightened by his scientific association, and encouraged by the counsels of his seniors, he develops a changing point of view. He begins to lose his rigidity of mind, to rely less on text books and more on his own observations, to have doubts, to develop an open mind, and above all to acquire a humility of thought—perhaps the greatest sign of wisdom. And then naturally come an assessment of the clinical value of his experiences; an examination of surgical procedures in the light of his developing judgement; an accumulation and assortment of these for comparison with others of a like nature in the future. Then follow speculations, inferences and hypotheses; the testing of these by further observations; and finally theories and discoveries. All these are milestones in the cultivated march of surgical judgement. If now Nature has been kind to him and his Christian and vocational roots lie deep, the end result should be the ideal surgeon.

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THE ROMAN HORACE — HIS HEALTH AND HIS POETRY

By Sir Gordon Gordon-Taylor, K.B.E., C.B., F.R.C.S., F.R.A.C.S.

IT is an honour and a great privilege to receive an invitation to contribute to the number of the *Australian and New Zealand Journal of Surgery* which is to be dedicated to my old friend, Sir Hugh Devine.

Surgery is a progressive science and art, and it would ill become one who has himself already attained octogenarian status to offer to the Editorial Committee an article concerned with the craft of surgery of which the recipient of this birthday number is such an acknowledged master.

I have therefore turned my gaze backwards two thousand years to the latter half of the century that preceded the birth of Christ, to the Augustan age of Rome, the era of Virgil, Tibullus, Propertius, Livy, Ovid and Horace, and have tentatively tried to probe the relationship, if any, between the poetry of Horace and the state of his health.

Quintus Horatius Flaccus was born on 8th December, B.C. 65, at Venusia in Apulia on the frontier of Lucania, and died on 27th November, B.C. 8, very shortly after the death of Maecenas, beside whom he was buried.

Kipling refers to the almost synchronous death of the two friends:

"Maecanas waits me on the Esquiline
Thither tonight go I . . .
And shall this dawn restore us, Virgil mine,
To dawn? Beneath what sky?"

The short communication which follows is the substance of an unpublished address to the Horatian Society at its Annual Dinner in the House of Lords on 21st July, 1958. A novel angle on the poet's work, a medical one, was introduced. The suggestion of Horace suffering from biliary disease may not be inappropriate in a surgical journal devoted on this occasion to Sir Hugh in view of his supreme gifts as an abdominal surgeon.

In my address I employed the following words:

"I feel somewhat self-conscious and very humble at finding myself in such an exalted position this evening. Alas! the indifferent health of some, or a previous important commitment on the part of others who had been approached for this occasion, has deprived Dr. Noble* of a really worthy *vis à vis*, nor has Death itself been idle, but has removed one who had promised to speak to us tonight.

Pallida mors aequo pulsat pede pauperum tabernas
Regumque turres.[1]

A replacement had to be found: the elderly victim who is "batting" second this evening bears little resemblance to the youthful hircine sacrifice promised to Fons Bandusiae.

cui frons turgida cornibus
primis et venerem et proelia destinat.[2]

Unfortunate me!

Fortuna saevo laeta negotio et
Transmutat incertos honores
Ludum insolentem ludere pertinax
Nunc mihi. [3]

I dare not contemplate what Horace would have thought of a surgeon participating at a festive dinner held in the poet's honour. Doubtless he would have angrily exclaimed:

Odi profanum vulgus et arceo;[4]
Favete linguis.

Nevertheless his indignation might have been lessened on hearing that a volume of his poems had been thumbed by that surgeon for more than seventy years, and that with a copy of the Odes in his pocket he had visited every continent in the world, even Russia, except South America.

*Peter Scott Noble, Principal of King's College, London.

1. Odes i. 3. 20.
2. Odes iii. 13. 4.
3. Odes iii. 29. 49.
4. Odes iii. 1. 1.

Although Horace could have known little of the benefits of surgery to mankind — in his day doubtless somewhat problematic — he was certainly aware of the value of surgery in forestry.

Duris ut ilex tonsa bipennibus
Nigrae feraci frondis in Algido
Per damna, per caedes, ab ipso
Ducit opes animumque ferro.[5]

Nor is this an inappropriate counsel for those charged with the treatment of many maladies, especially cancer, at this present day, two thousand years after these lines were first written.

I have for many years listened admiringly to the now lengthy saga of speakers at this dinner, a group which has included statesmen, proconsuls, ambassadors, public orators of ancient universities, Her Majesty's Judges and other distinguished public figures, and have come to realize how difficult it is to break fresh ground in presenting Horace to you — as difficult as with our biennial Oration at the Royal College of Surgeons commemorating our patron Saint, John Hunter (1728-93) who is interred in Westminster Abbey.

Tonight I want to speculate on the possibility of a relationship between the state of Horace's health and his physical condition on the one hand and on the other some of those wistful lines of retrospect and also what one commentator has called "the plangent undertones" of some of the poet's verse "like the sound of an iron bell announcing the universal doom." I am a trifle dubious about the propriety of introducing the subject of ill health on a convivial occasion such as this when we have enjoyed:

mero superbo
pontificum potiore cenis.[6]

In endeavouring to elaborate this theme I am naturally thinking of such lines as:

Omnes eodem cogimur, etc.[7]

or:

Lusisti satis, edisti satis atque bibisti
Tempus abire tibi est.[8]

or:

tamen illic vivere vellem
Oblitusque meorum, obliviscendus et illis[9]

or the wonderful *Ode to Postumus*[10] with its

Labuntur anni nec pietas moram
Rugis et instanti senectae
afferet indomitaeque morti,

and:

Linquenda tellus et domus et placens
Uxor,

or the famous *Diffugere Nives*[11] with its pathetic *Pulvis et umbra sumus* and:

virtus recludens immeritis mori[12] *caelum.*

But one might go on indefinitely.

No one could ever have expected jocose comedy from an author like Gibbon, tormented by an enormous genital encumbrance which was of record size and which ultimately killed him. We look in vain for charm of subject or of touch in an author like Guy de Maupassant who was to die from cerebral syphilis and was already infected with the taint when he wrote *Boule de Suif* and *Le Horla*. The composition of a Dictionary seems appropriate to the corporal magnitude and the gall-stones of Samuel Johnson. It was only the sense of honour of Sir Walter Scott in his endeavours to satisfy his creditors that enabled him to give to the world a succession of historical and other novels, even during the darkest days of his attacks of biliary colic (1817-19) — in particular the *Bride of Lammermuir* which some have even thought his best was the product of a drugged and abnormal condition. One might well have expected Robert Louis Stevenson's writings to be coloured by his long personal experience of illness, but with his wings imped by the proverbial *spes pthisicorum* and his own nomadic spirit he was able to travel hopefully and captivate a generation. The affliction of Milton and of Axel Munthe is reflected in the produce of their pen.

By and large it is true that *l'horizon d'un auteur se rembrunit par sa mauvaise santé:* it is difficult to write happily at all times, with an eagle tearing at one's viscera and vitals.

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5. Odes iv. 4. 57.
6. Odes ii. 14. 28.
7. Odes ii. 3. 28.
8. Epist. ii. 2. 214.
9. Epist. i. II. 9.
10. Odes ii. 14. 4.
11. Odes iv. 7. 1.
12. Odes iii. 2. 21.

In a recent work on Horace it is stated that on the whole Horace enjoyed excellent health. But did he? He was *never*, at any rate after his early manhood, a man in rude health. In Epistles 1, 7, written before B.C. 21, when he was only 44 years of age or under, he writes to Maecenas who had apparently reproached him for staying in the country longer than he had said he would:

Si me vivere vis sanum recteque valentem
Quam mihi das aegro, dabis aegrotare timenti,
Maecenas, veniam.[13]

and in the same Epistle he declares his intention of going to the seaside in the event of the snows of winter "lying on the Alban fields" and only returning to Rome with the zephyrs and the first swallow.

Quod si bruma nives Albanis illinet agris,
Ad mare descendet vates tuus et sibi parcet
Contractusque leget.[14]

Sibi parcet! Will be "careful of himself," though only in his early forties!

In the third epode written about B.C. 30, when about 35 years of age, there is an account of the severe attack of pain in the upper part of the abdomen and shoulder for which he blames garlic.

Quid hoc veneni saevit in praecordiis? . . .
Nec munus umeris efficacis Herculis
inarsit aestuosius.[15]

With regard to the use of *umeris* where scapula might have been expected by surgeons of today, the former word was often employed for the shoulder region, and in any case the word is found more frequently than scapula in the literature of Republican and Augustan Rome. Incidentally, I have been informed by medical guests as well as by patients that garlic or onions often precipitate discomfort in those with gall-stones or any disorder of the gall-bladder.

Was then Horace a fellow sufferer from gall-stones with Sir Walter Scott, Montaigne and Samuel Johnson? His own description of himself is compatible with such a diagnosis.

Me pinguem et nitidum bene curata cute vises,[16]
Cum ridere voles, Epicuri de grege porcum.

Suetonius tells us that Horace was *brevis atque obesus* and quotes a joke of Augustus *à propos* his figure, *Vereri autem mihi videris ne maiores libelli tui sint quam ipse es; sed tibi statura deest, corpusculum non deest.* Augustus even jokingly calls him *sextariolus*, a pintpot.

"Fair, fat and forty" — the gall-stone type. Although Horace tells us that he had black eyes and at one time black hair, he subsequently became prematurely grey. Not necessarily, he may have been quick tempered, but that may have only been the Latin characteristic.

His appreciation of good food and wine is well-known from his writings, and those of us who were privileged to be present at the Bimillenary Dinner of the Society in 1935 had their memories refreshed as to his fancies. When only 35 years old he sighs for the simple life and good health (Sat 11, 1, 70). "Now learn what and how great are the blessings that simple living brings in its train. Firstly, good health, for how harmful to a man a variety of dishes is you may realise if you recall the plain food which agreed with you in former days."

The prescription[18] for the jaded boon companion to enable him to continue drinking would seem to promise every manner of aftermath, and if the *convivae* did not develop gall-stones or some disease of the biliary tract they must have been under the special protection of Heaven.

Horace also refers to his own dyspepsia in the Satire describing the journey to Brundisium,[19] excusing his retirement to sleep rather than playing ball like Maecenas. His indigestion occasions little surprise when he writes on bathing with stomach gorged with food.

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13. Epist. i. 7. 3.
14. Epist. i. 7. 9.
15. Epod. iii. 5 and 17.
16. Epist. i. 4. 16.
17. Ars. Poet 37.
18. Sat. ii. 4. 58.
19. Sat. i. 5. 49.

Crudi tumidique lavemur.[20]

What of gall-stones? You may say. Are not gall-stones a modern malady? They are as old as the hills and were certainly present in a mummy of a priestess of the XXI Egyptian Dynasty (circa 1500 B.C.) which we possessed at the Royal College of Surgeons till it was destroyed by enemy air attack in 1941. But apart from dietetic abuses or orgies the climatic conditions of Rome with endemic typhoid and paratyphoid infections and with his health doubtless impaired by malaria from the Pontine Marches may easily have aged him prematurely and conditioned his writings.

But there is other evidence of early senescence. In the Fourth Book of the Odes which appeared in B.C. 13 when he was only 52, there is this cry:

Non sum qualis eram bonae[21]
sub regno Cinarae.

and again in his invitation to Phyllis, the *psaltria*, to be his guest in his home:

meorum finis amorum
Non enim posthac alia calebo femina.[22]

Men have chuckled at Voltaire's excuses to his mistress, Madame du Chatelet, when he was only forty-six years of age, and after all an action for paternity was brought against a nonagenarian Chancellor of a Commonwealth University: of the success of the action I know not, but the Chancellor was hugely flattered.

We are left with the impression of an ageing Horace. I cannot think that Horace committed suicide despite the rapidity with which his death followed Maecenas'. To those accustomed to weigh signs and symptoms Horace was already exhibiting signs of physical deterioration, and some complication of disease of the biliary tract or of a raised blood pressure doubtless carried him off.

I have devoted my time to one particular facet of the Horatian tradition, but some of his philosophy I am glad to have adopted for my own purposes at all times:

Quid sit futurum eras, fuge quaerere et[23]
Quem fors dierum cumque dabit, lucro
Appone, nec dulces amores
*Sperne, puer, neque tu choreas.**

and especially those lines from the Poet's Prayer.**

Frui paratis et valido mihi[24]
Latoe, dones, at precor integra
Cum mente, nec turpem senectam
Degere nec cithara carentem

and finally

Dulce est desipere in loco.[25]

The background of Rome against which this *scenario* is set may not be unbefitting in the case of the great surgeon whom we desire to honour and whose faith inevitably directs his gaze to the Eternal City on the Tiber, while the *lingua Latina* which now and then finds its way into the text will not be unfamiliar to one the ritual of whose religion perpetuates from time immemorial perhaps the greatest and grandest language of all time.

The Latin tongue, too, reminds the writer of that famous hospital in Melbourne which Sir Hugh has served so devotedly, an institution ministered to by members of that religious faith that has helped him to withstand on more than one occasion the hammer blows of fate, and to rise with head unbowed, beloved and respected by the surgical profession per *orbem terrarum*.

*As not your future fate — enjoy
Each day as so much gain,
And don't despise the girls, my boy,
Nor think the dance is vain.

**My Prayer,
Son of Latona, is no more
Than to enjoy my frugal store
Sound both in body and in mind;
Nor as old age steals on, to find
My harp unstrung, or friends unkind.

20. Epist. i. 6. 61.
21. Odes iv. 1. 3.
22. Odes iv. 11. 33.
23. Odes i. 9. 13.
24. Odes i. 31. 17.
25. Odes iv. 12. 28.

PULSION DIVERTICULA OF THE ESOPHAGUS AND THEIR TREATMENT

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INTRODUCTION

IN most persons the esophagus, or gullet, functions quietly and efficiently from birth to death. Indeed, it so usually functions well that its dysfunctions, when they occur, are often neglected or minimized by both patients and physicians. Yet the esophagus can be the site of a variety of diseases and lesions which may have serious consequences if neglected but which can be treated successfully by appropriate surgical means. Among the more common lesions responsible for esophageal dysfunction are esophageal diverticula, and they offer a particularly rewarding opportunity for effective surgical treatment.

Differentiation from traction diverticula

It is generally agreed that diverticula of the esophagus can be divided into two general types: pulsion diverticula and traction diverticula. Traction diverticula usually are located in the middle third of the esophagus. They probably result from some inflammatory disease of the lymph nodes around the trachea and major bronchi. The inflamed nodes become adherent to the outer wall of the esophagus; and as the inflammatory process subsides and the swollen node contracts, it pulls the esophageal wall outward, producing a diverticulum. Ordinarily such a diverticulum has a rather large mouth and empties itself freely, so that there is no retention of esophageal contents. It rarely causes symptoms and usually is discovered incidentally during roentgenologic examination of the esophagus and stomach. Tracheo-esophageal or broncho-esophageal fistula can develop in association with traction diverticula, and bleeding from ulceration of such diverticula has been reported. Such complications of these diverticula are uncommon, however; and treatment of the diverticula is not often necessary.

Pulsion diverticula of the esophagus, on the other hand, frequently produce symptoms that require relief. This can best be accomplished in most instances by surgical excision of the diverticula.

FIG. 1. Roentgenologic appearance of aspiration pneumonitis due to pharyngo-esophageal diverticulum.

ORIGIN, DEVELOPMENT AND DIAGNOSIS

The sites at which pulsion diverticula devclop are remarkably constant. They arise at the posterior wall of the junction of the pharynx and the cervical esophagus or in the lateral wall of the lower third of the esophagus. The former are commonly called pharyngo-esophageal diverticula and the latter epiphrenic or supradiaphragmatic diverticula of the esophagus.

Beginning and etiology

The average age of patients presenting themselves with symptoms resulting from pulsion diverticula of the esophagus is approximately 55 years. (The youngest patient I

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personally have seen with a pulsion diverticulum of the esophagus was 34 years of age.) This fact, of course, indicates that these are acquired and not congenital lesions. On the other hand, the very constant site of origin of pulsion diverticula, either at the posterior wall of the pharyngo-esophageal junction or in the lateral wall of the lower thoracic zone of the esophagus, indicates presence of congenitally weak points there in the muscular layers of some individuals which permit the development of diverticula.

occur after eating. A noisy gurgling in the throat often accompanies the act of swallowing and may be a source of considerable embarrassment. There is a sensation of fullness in the throat that sometimes can be relieved by pressure applied to the side of the neck at about the level of the thyroid gland.

The rather rapid increase in the size of the established diverticulum is due to both the recurrent pressure involved in the act of swallowing and the more or less constant

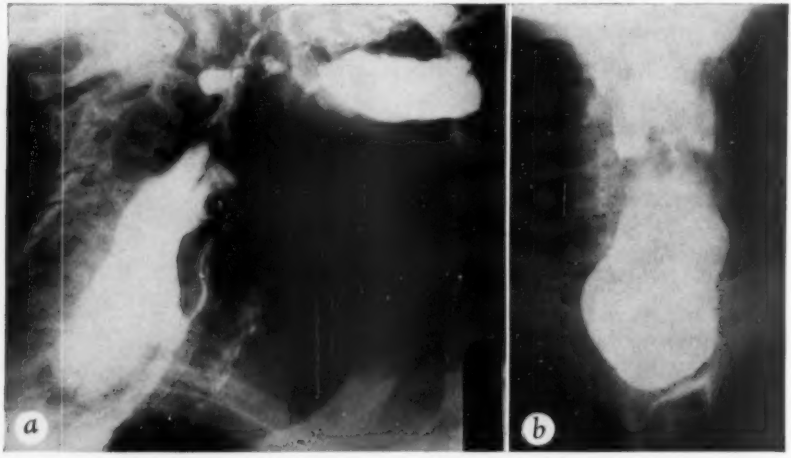

FIG. II. (a) Lateral view of large pharyngo-esophageal diverticulum. (b) Antero-posterior view of large pharyngo-esophageal diverticulum.

Beyond this, the etiology of pulsion diverticula seems quite clear. The act of swallowing results in pressure against the walls of the esophagus. At a point where the muscular wall is congenitally weak or deficient this pressure causes the esophageal mucosa to herniate. Gradually over a period of years a pouch of herniated esophageal mucosa develops and a diverticulum is formed. Once the diverticulum has developed, it tends to increase in size at an accelerating rate and to produce symptoms of increasing severity.

Symptoms and growth

The earliest symptom of a pharyngo-esophageal diverticulum is usually the sensation that a foreign body is obstructing the normal process of swallowing so that food seems to catch in the throat. Later, regurgitation of undigested food and mucus may

distention by retained food and secretions. The sac ordinarily will extend downward between the esophagus and the prevertebral fascia into the mediastinum. Usually it tends toward the left of the midline as it descends, but occasionally it is more prominent on the right. As it increases in size and descends, the diverticulum tends to angulate the esophagus, which is obstructed both by this angulation and by pressure of the sac against the posterior wall of the esophagus. Eating becomes a slow, laborious process. Severe coughing and choking spells may accompany the act of swallowing, because of regurgitation of the contents of the diverticulum into the trachea. Loss of weight and malnutrition may occur. Aspiration pneumonitis and lung abscess may be very serious complications of a neglected pharyngo-esophageal diverticulum (Fig. I).

The symptoms of epiphrenic or supradiaphragmatic diverticula are similar to those of the pharyngo-esophageal lesions except that dysphagia occurs at a slightly later phase in the act of swallowing and the sensation of fullness is located in the thorax instead of the neck. In general, the symptoms of diverticula of the lower thoracic esophagus tend to be less severe and to worsen less rapidly than those due to pharyngo-esophageal diverticula.

Associated conditions and esophagoscopy

The presence of a pulsion diverticulum of the esophagus can be strongly suspected from the patient's symptoms. The diagnosis is readily established by appropriate roentgenologic examinations (Fig. IIa and b). Esophagoscopy is not usually necessary in patients with pharyngo-esophageal diverticula, and indeed it should not be performed in the ordinary case because of the danger of esophageal injury. Occasionally, if the clinical symptoms and roentgenologic examinations are not entirely typical and definite, this measure may be indicated to determine the presence or absence of some other esophageal disorder. Great care is essential, however, in performing esophagoscopy in the presence of a pharyngo-esophageal diverticulum.

In most instances of diverticulum of the lower thoracic esophagus, esophagoscopy should be carried out. Experience here has shown that many of these diverticula are associated with achalasia of the esophagus or esophageal hiatal hernia with esophagitis; and it is important to investigate this possibility so that appropriate treatment for these associated conditions, if indicated, can be carried out in combination with that for the diverticula. Often it is wise to perform esophageal dilation before operation for esophageal diverticulum, to make sure there is no other obstruction of the esophageal lumen. This can be accomplished easily and safely only if a previously swallowed silk thread is used as a guide for the dilating bougie.

TREATMENT FOR PHARYNGO-ESOPHAGEAL DIVERTICULA

History

Pharyngo-esophageal diverticula were first recognized almost two hundred years ago. Although it was apparent even then that these lesions could not be treated effectively except by surgical means, surgical methods for this purpose were slow to develop. After some attempts by others, von Bergmann, in 1892, reported the first successful operation for this condition. Early operations were associated with a high mortality due to mediastinitis and pulmonary complications; and even in patients who survived, the morbidity due to esophageal fistula was great. In order to deal with these complications, Goldmann, in 1909, introduced the two-stage operation for these diverticula. This procedure gained wide acceptance, and even as recently as a few years ago Lahey (1954) strongly advocated the two-stage operation as the procedure of choice. Other surgeons, notably Harrington (1939) and Sweet (1956), however, developed one-stage operations which have proved to be just as safe and satisfactory as two-stage operations for pharyngo-esophageal diverticula, and which have definite advantages to be discussed later. At present there can be few if any indications for a two-stage attack on pharyngo-esophageal diverticula.

Indications and preparation

The indications for one-stage operation are clear. Very small diverticula produce no symptoms of consequence. Any diverticulum large enough to produce symptoms sufficient for diagnosis is large enough to warrant operation for its removal.

Proper preparation for operation is essential. If the patient is very poorly nourished or dehydrated, or has aspiration pneumonitis or a lung abscess, a considerable period of pre-operative treatment and preparation may be necessary. Rarely, it may be advisable to establish a gastric stoma for feeding purposes if the situation has been badly neglected. As mentioned previously, if there is any evidence of obstruction or angulation of the esophagus, it should be dilated before operation, with use of a previously swallowed thread as a guide for the dilating bougie. Always, to avoid aspiration of unwanted material into the respiratory tract during the operation, the diverticulum should be emptied of all retained secretions, food, or barium before anesthesia is induced.

Anesthesia and approach

Both local and general anesthesia have been used for these operations. In our own earlier

experience, my colleagues and I used cervical block anesthesia extensively and found it satisfactory on the whole. However, surgical procedures under local anesthesia are not well tolerated by some nervous, apprehensive patients; so more recently general anesthesia with an endotracheal tube in place has been used in most cases.

As a rule, the diverticulum can be recognized promptly, arising from the posterior wall of the pharyngo-esophageal junction at a point a little above the level of the omohyoid muscle. The sac extends downward, posterior to the esophagus. If operation is performed under local anesthesia the patient can be asked to swallow, and the

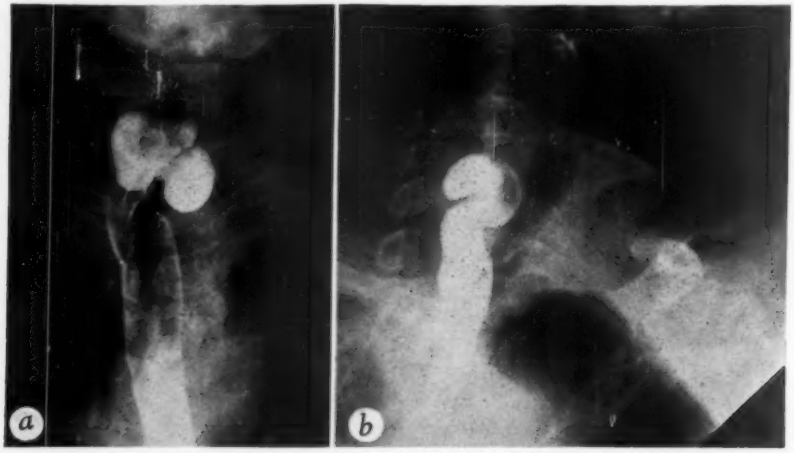

FIG. III. (a) Unusual lateral origin of pharyngo-esophageal diverticulum as seen in postero-anterior view. (b) Lateral view of unusual type of pharyngo-esophageal diverticulum.

Some surgeons have advocated a modified thyroidectomy incision or a skin crease incision for the surgical approach to pharyngo-esophageal diverticula, asserting an advantage in cosmetic result. My colleagues and I have found that an oblique incision along the anterior border of the sternocleidomastoid muscle offers better exposure and that the scars from this incision are quite satisfactory cosmetically. Although a pharyngo-esophageal diverticulum can be excised from either a right-sided or a left-sided cervical incision, we most often have made the approach from the left because most diverticula extend slightly to the left. The skin incision along the anterior border of the sternocleidomastoid muscle extends from the hyoid bone to a point about an inch above the clavicle. This muscle is retracted laterally and the incision is deepened, exposing the omohyoid muscle. The carotid sheath and its contents are retracted laterally, and the thyroid gland and larynx are retracted medially.

bulging of the diverticulum with this act will aid precise identification.

Excision and closure

The diverticulum is grasped with an Allis forceps and is elevated into the wound. Dissection is carried down to the neck of the sac and its site of origin. It must be recognized that variations in the site of origin of these diverticula can occur, and that occasionally lateral pharyngeal diverticula may occur (Fig. IIIa and b). Dissection of the neck of the diverticulum must be performed carefully to avoid injury to the recurrent laryngeal nerve, which is close to it in many instances. Excision of the diverticulum must be complete, so that no small sac is left to initiate a recurrence. The muscle and fascial layers are carefully dissected back until only the mucosa remains.

Various technics for closure and repair have been described and used successfully. My own preference is to place a curved clamp

across the neck of the sac. The clamp has a pin in its distal end to prevent the diverticulum from slipping out of it. With the clamp serving as a tractor to hold the diverticulum in position, the mucosa of the esophagus is incised proximal to the clamp and then closed with interrupted silk sutures, the knots being placed inside the lumen of the esophagus and some of the sutures left long for use as tractors (Fig. IVa). After the mucosal closure is completed and the diverticulum removed, the muscular and fascial layers of the esophagus are closed in a transverse manner using a fine continuous catgut suture (Fig. IVb). This technic accomplishes an accurate, complete removal of the diverticulum and avoids any danger of narrowing or stricture of the esophagus at the site of repair. A small rubber drain is placed down to the site of esophageal closure and the platysma and skin are closed with interrupted sutures.

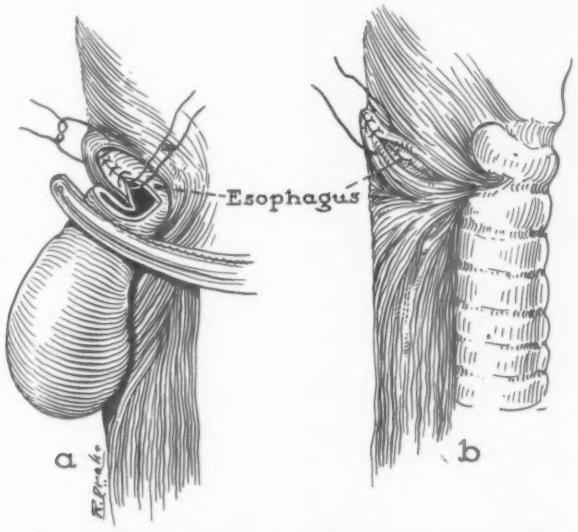

FIG. IV. Technic of one-stage excision of pharyngo-esophageal diverticulum. (a) Clamping and excision, with suturing of mucosa. (b) Suturing of muscular and fascial layers.

Post-operative care and recovery

The results of operation with this technic have been very satisfactory. It has not been necessary to insert a tube for temporary feeding purposes. The patient is allowed to begin to swallow small amounts of water twenty-four to thirty hours after operation. Gradually the liquid and solid intake is increased. The rubber drain is removed on the third post-operative day. Most patients leave the hospital by the fifth post-operative day, and by then are able to eat soft foods without difficulty. The skin sutures are removed on the sixth post-operative day, at which time most patients are dismissed. They are advised to chew their food well and to omit rough or bulky foods for about two weeks. Routine use of antibiotics is not necessary in the post-operative management of these patients, and occasions for administration of them are rare.

Evaluation of method

Although even in recent years some surgeons have advocated a two-stage operation as preferable to a one-stage operation for pharyngo-esophageal diverticula, there seems to be little evidence to support this view. The two-stage operation requires two anesthetics and a much longer period of hospitalization, and incurs a greater risk of complications such as fistula and injury to the recurrent nerve. The only advantage of a two-stage operation is that the time between stages allows the cervical fascial planes to

become walled off so that if leakage or infection occurs it is localized and does not spread into the mediastinum. Actually, if a one-stage operation is performed properly there is very little chance that mediastinitis will occur; and if it does occur the drain inserted at the time of operation provides adequate treatment. The approach used in excising a pharyngo-esophageal diverticulum is exactly the incision that would be used to treat cervical mediastinitis due to some other

FIG. V. Large diverticulum of lower thoracic esophagus with associated esophageal hiatal hernia.

cause, such as lodgment of a foreign body or endoscopic perforation of the cervical esophagus; and, in fact, effective treatment of cervical mediastinitis can be accomplished by the one-stage operation. Evidence of the applicability and effectiveness of one-stage operation for pharyngo-esophageal diverticula is the experience of the Mayo Clinic. One-stage operations have been performed in every case without exception for the past twenty-five years. In the period from January, 1944, through December, 1957, 478 one-stage operations were performed.

TREATMENT FOR EPIPHRENIC DIVERTICULA

Incidence, indications and dimensions

Pulsion diverticula do not occur so frequently in the lower thoracic esophagus as in the cervical zone, but they are not rare. During the ten-year period from 1944 through 1953, this lesion was demonstrated radiologically in 201 patients at the Mayo Clinic. As mentioned previously, the symptoms of these diverticula are similar to those of pharyngo-esophageal diverticula but are usually not so severe. Actually, only 24 of the 201 patients with this lesion had enough symptoms to warrant operation; and it is worthy of note that in 17 of these 24 patients there were other abnormal conditions involving the lower esophagus, such as hiatal hernia (Fig. V), cardiospasm (Fig. VI), diffuse spasm of the esophagus, and esophagitis, which seemed to contribute to the symptoms noted.

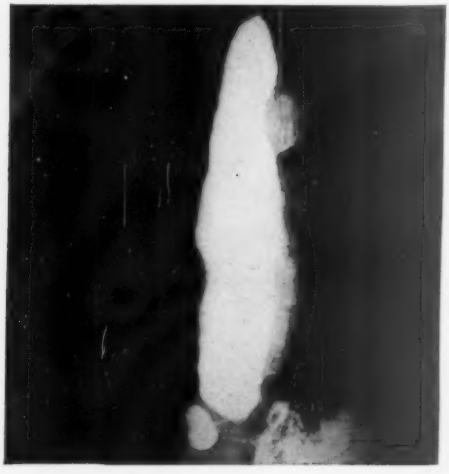

FIG. VI. Small diverticulum of lower thoracic esophagus with associated achalasia of the esophagus.

My colleagues and I are firmly of the opinion that operation for pulsion diverticula of the lower thoracic esophagus is indicated only if the symptoms are progressive and severe. The presence of the defect with few or no symptoms does not warrant operation.

We have been able to observe a number of patients with pulsion diverticula of the lower thoracic esophagus over a period of years, and only in relatively few did the development of symptoms require operation. In most of these some other associated condition involving this anatomic region was present or developed. The major symptoms of those patients coming to operation were dysphagia, regurgitation, vomiting, aspiration and cough, loss of weight, and substernal distress. This group averaged 53 years of age, and the duration of symptoms varied from a few months to thirty years with an average of about five years.

Pulsion diverticula of the lower esophagus present to the right about twice as frequently as to the left. They usually occur 4 to 8 cm. above the esophagogastric junction. Rarely, multiple diverticula occur (Fig. VII). Radiologically, they appear as globular sacs arising from the lower esophagus with necks of various widths. The size of the diverticula seen here has varied from 2 cm. to 11 cm. The necks varied from 1.5 to 4 cm. in width and usually were less than 1 cm. long.

Surgical measures

Esophagoscopy should be carried out in all patients considered for operation, so that any associated lesions involving the esophagus can be identified. Dilation of the esophagus by passage of a sound over a previously swallowed thread should be a routine preoperative measure.

Right or left thoracotomy is performed, depending on the position of the diverticulum and the nature of any associated lesion involving the lower esophagus. (For example, left thoracotomy is preferred if there is an associated hiatal hernia, even though the diverticulum presents on the right.) The esophagus and diverticulum are mobilized carefully and dissection is extended down to the neck of the diverticulum, which protrudes through a smooth oval defect in the muscular layers of the esophagus (Fig. VIIIa). The sac is excised completely, but with care not to narrow the lumen of the esophagus. The mucosa is closed with fine interrupted sutures of silk (Fig. VIIIb). The muscular layers are closed with a continuous suture of fine catgut and, if feasible, the mediastinal pleura is used to seal the site of repair (Fig. VIIIc). A gastric tube is passed through the nose into the esophagus and stomach.

FIG. VII. Multiple (two) large diverticula of the lower thoracic esophagus with associated small esophageal hiatal hernia.

It is extremely important that any associated lesion be treated at the same time the diverticulum is excised. If there is an associated hiatal hernia it is repaired. If there is associated diffuse spasm of the esophagus, a fairly extensive longitudinal myotomy of the involved area is performed. A cardiomyotomy of the Heller type is performed if there is cardiospasm.

Closed drainage of the pleural space is established and the chest wall is closed. The gastric tube can be removed in twenty-four to forty-eight hours and fluids are administered orally in gradually increasing quantities after this time. Most patients should be able to eat a soft diet and leave the hospital in approximately a week.

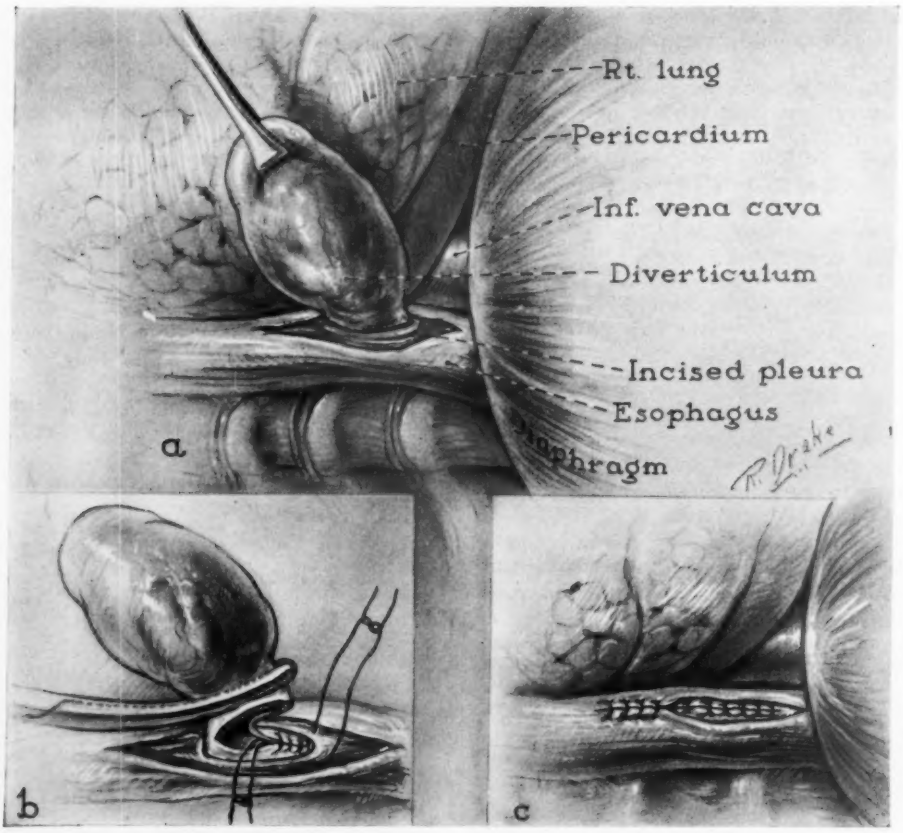

FIG. VIII. Technic of excision of diverticula of the lower thoracic esophagus. (a) Mobilization of diverticulum and exposure of its neck. (b) Closure of mucosa. (c) Closure of muscular layers. (Reproduced, with permission of the publisher, from: Habein, H. C., Jr., Kirklin, J. W., Clagett, O. T. and Moersch, H. J.: "Surgical Treatment of Lower Esophageal Pulsion Diverticula." *Arch. Surg. (Chicago)*, (1956), vol. 72, page 1018.

COMMENT

Pulsion diverticula of the esophagus occur not infrequently. They produce symptoms that are annoying and embarrassing, and can give rise to complications actually endangering the patient's life. But diagnosis of them is not difficult, and surgical means for treating them are effective with little risk.

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SURGICAL CONSIDERATIONS IN HIATUS HERNIA

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THERE are still many problems to be resolved in the understanding of the aetiology, pathology and treatment of this increasingly frequent and severe disability. There is no doubt that a large number of people with this complaint require surgical treatment, but there is unfortunately still no surgical agreement on the best method of approach to the problem. Because of fundamentally differing approaches, surgical views will remain irreconciliable till more precise knowledge of the aetiology, pathological physiology and results of treatment have become known.

Many surgeons will argue that hiatus hernia is best treated by operation from below the diaphragm, while others, not only thoracic surgeons, maintain that operation from above the diaphragm offers the best guarantee of a permanent reduction. It is the purpose of this paper to look at the problem from both points of view, to point out the advantages and disadvantages of each, but more particularly to emphasize some of the problems in diagnosis and to suggest that careful attention to anatomical detail in repair offers to the surgeon the best guarantee of a permanent reduction either from above or below.

CLASSIFICATION OF HIATUS HERNIA

Following on the illuminating work of Allison (1948, 1951, 1953), three principal varieties of herniation of the stomach through the oesophageal hiatus are recognized.

In the first the gastro-oesophageal junction has risen into the posterior mediastinum and the portion of stomach lying in the thorax has carried up with it a pouch of peritoneum. This pouch lies anteriorly and laterally but not posteriorly, as many illustrations would suggest (see Fig. I). This figure illustrates the peritoneal pouch drawn up anteriorly and the absence of such a pouch posteriorly. To this type of hernia the word "sliding" has been applied. This term has led to some misinterpretation clinically and radiologically. It referred originally to the way the gastro-oesophageal junction slid up into the mediastinum. It also had an anatomical significance analogous to the inguinal hernia, implying an incompletely formed sac. Finally, to many minds, it has an active significance, i.e., in this type of hernia the stomach is constantly sliding up and down in the posterior mediastinum.

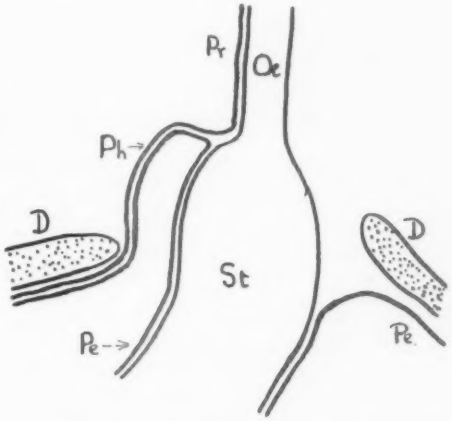

FIG. I. Diagrammatic representation of a hiatus hernia showing the absence of the peritoneal pouch posteriorly.
Oe—oesophagus. St—stomach. D—diaphragm. Pr—fascia propria of oesophagus. Ph—phrenico-oesophageal ligament blending with fascia propria. Pe—peritoneum.

Operations on hitaus hernia from above have shown that in a large percentage of cases this could not happen. As a result of oesophagitis and peri-oesophagitis in many cases and also less commonly changes in the gastric pouch, adhesions form between the oesophagus and peri-oesophageal tissues and pleura; the sac wall often becomes adherent to the pleura or pericardium and adhesions occur within the sac. Such herniae are

in fact irreducible and no longer sliding. Further confusion arises as a result of radiological studies when a "sliding" hernia is "made to appear" in the Trendelenberg position. In many cases this does occur, but also in many others the appearances are due to the filling out of a hernia already present and often irreducible.

the same ominous significance, as regurgitation is not a complication. Many such patients with large irreducible herniae may have no symptoms whatever and are discovered commonly by routine chest X-ray.

The final group comprises those combining the features of both of the previous groups.

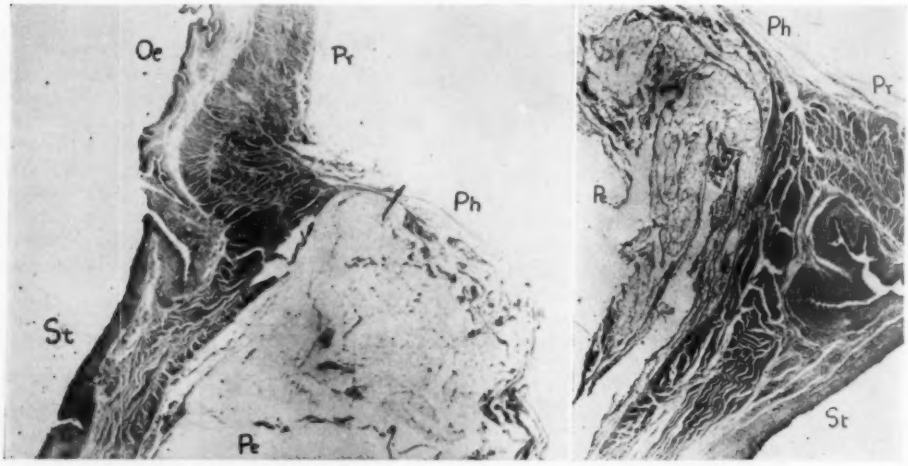

FIG. II. Moderate and high power photomicrographs of vertical sections through oesophago-gastric junction in a hiatus hernia. This shows the oesophagus above and stomach wall below, the junction recognizable by mucosal change. The phreno-oesophageal ligament is seen blending with fascia propria and separated from peritoneum by a collection of fat.

Oe—oesophagus. Pe—peritoneum. Ph—phreno-oesophageal ligament. Pr—fascia propria. St—stomach.

This confusion has led to some incorrect thinking in regard to treatment. It is argued that if a hernia becomes obvious on adoption of the Trendelenberg position then the hernia can be kept reduced by instructing the patient to sleep propped up. This indeed may be quite untrue and has led to unjustified conservatism in the treatment of irreducible hiatal herniae of this type. It is these herniae which are most commonly associated with persisting though lessened symptoms of regurgitation on medical management and in whom prolonged delay before surgery leads to rapidly diminishing chances of effecting permanent surgical reduction.

The second type described by Allison, the para-oesophageal hernia, implies that portion of the stomach has "rolled" up into the preformed peritoneal pouch anterior and lateral to the oesophagus while the gastro-oesophageal junction remains in the normal situation. These may be reducible or irreducible, but in these irreducibility has not

This state of affairs is arrived at either by a sliding hernial sac becoming larger by further portions of the stomach rolling up into it or, conversely, by the para-oesophageal hernia becoming so large that it interferes with the crura and attachments of the gastro-oesophageal junction which subsequently rises into the mediastinum.

Our attention in this paper will be directed mostly towards the sliding hiatal hernia.

SURGICAL ANATOMY

In our present state of knowledge we recognize two principle factors responsible for the retention of the oesophago-gastric junction in its normal position.

(a) Muscular tone and integrity of the right crus.

(b) Attachment to the oesophago-gastric junction of the phreno-oesophageal ligament, extraperitoneal tissues and

peritoneum of which the first is the most important.

A third factor—

(c) The length of the gastric vessels—has been suggested, but considerable doubt exists as to the importance of this.

Fig. II is a photomicrograph of the oesophago-gastric junction taken from a woman of 65 who died of a cerebro-vascular accident and who was found at post-mortem to have a small sliding hernia. The pleura has been removed in the preparation of the specimen. It shows the sac wall comprising

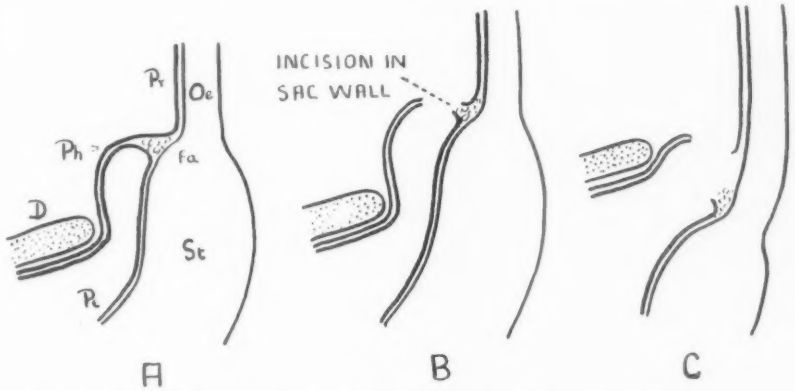

FIG. III. Diagram of hiatus hernia A. B shows site of incision through sac wall in operation from above. C shows separation of phreno-oesophageal ligament and peritoneum and loss of definition of ligament on reduction. Oe—oesophagus. St—stomach. Fa—fat. D—diaphragm. Ph—phreno oesophageal ligament. Pr—fascia propria of oesophagus. Pe—peritoneum.

As a result of age, obesity and many other causes of increased intra-abdominal pressure, such as repeated pregnancies, the factors above are so interfered with that herniation occurs. The aim of surgical treatment is to secure complete and permanent reduction of the gastro-oesophageal junction below the diaphragm, and this aim can only be achieved surgically by careful attention to the restoration of both factors. The simple approximation of crural fibres and a few stitches in the stomach will not suffice to prevent recurrence.

As was mentioned earlier when the stomach rises into the mediastinum it carries with it a pouch of peritoneum lying anteriorly and laterally and with this pouch the phreno-oesophageal ligament is stretched and forms part of the sac wall (see Fig. II). Many people doubt the existence of this ligament, which is a condensation of deep fascia passing from the under surface of the diaphragm to the region of the oesophago-gastric junction, there blending with the fascia propria of the oesophagus. It is covered on the thoracic surface by pleura and on the abdominal surface is separated from the peritoneum by a variable amount of extraperitoneal fat.

peritoneum, extraperitoneal fat and phreno-oesophageal ligament. The ligament can be seen passing to the oesophago-gastric junction, recognized by the mucosal change and there blending with the fascia propria of the oesophagus.

It is important to realize that only anteriorly and laterally can the association of ligament and peritoneum be satisfactorily demonstrated and a satisfactory fringe obtained for suture to the diaphragm. Posteriorly the ligament cannot be demonstrated nor does a peritoneal pouch develop, for here stomach and diaphragmatic muscle are in intimate contact over a broad area and no condensation of extraperitoneal issue to form a ligament is necessary.

Anteriorly however, the stomach and oesophagus are separated from a rim of diaphragmatic muscle by an interval and the only way of preserving their normal relationship to the hiatus is by the development of a ligament. The important surgical consideration arising out of this is that careless dissection in the front may destroy the attachments of the ligament and peritoneum to the

oesophago-gastric junction and so remove one of the important mechanisms available to the surgeon for securing permanent reduction. Fig. XVI is designed to show how in the operation from below after the hernia has been reduced an incision through the peritoneum and ligament can be made to secure a satisfactory fringe of tissue for suture.

The phreno-oesophageal ligament and peritoneum are not adherent to one another and can be easily separated. This has surgical importance in the operation from above the diaphragm. If the sac wall comprising ligament and peritoneum is divided close to the oesophago-gastric junction and then the stomach pulled down, peritoneum and ligament are separated and in fact the oesophageal end of the ligament may remain in the chest disappearing into the fascia propria (see Fig. III). This can be avoided by the insertion of sutures before reduction is performed, as will be explained later.

The main factor in the prevention of herniation of the stomach posteriorly through the hiatus is the integrity of the crura. Studies on the anatomy of the crura are numerous and reference should be made to Carey and Hollingshead (1955) and Collis *et alii* (1954). These writers have shown adequately how the hitaus is formed by the separation of the fibres of the right crus into two limbs. The orifice becomes larger by the stretching of the two limbs, and when the hernia is reduced a large gap will be found posteriorly. Shortening of the crural limbs and obliteration of the posterior gap is achieved by loose approximation of the two limbs posteriorly.

PHYSIOLOGICAL CONSIDERATIONS

The problem of why the cardia is competent has not yet been solved, but there has been a recent change in thought away from the concept of extrinsic factors such as the action of crural musculature and preservation of the acute angle between stomach and oesophagus, to a concept of intrinsic mechanisms.

From oesophageal pressure tracings there is now no doubt concerning the existence of a zone of high pressure at the lower end of the oesophagus acting as a physiological sphincter. This is seen also on barium studies and does not correspond to the position of the crura. This physiological sphincter action at the lower end forms one of the barriers to regurgitation. The second recent contribution is that of Botha (1958) who supposes a mucosal plugging action at the oesophago-gastric junction, the redundant mucosa acting as a valve preventing regurgitation. This mucosal activity is carried on by the muscularis mucosae.

The mechanisms responsible for competence fail for reasons that are at present unknown. They may fail under physiological conditions of increased intra-abdominal pressure such as pregnancy when no hernia can be demonstrated. They may fail in some patients in whom no cause of increased intra-abdominal pressure exists, nor in whom can a hernia be demonstrated. In these patients increased intra-gastric pressure from increased secretion or air swallowing may be the answer. Conversely the mechanisms of competence may be completely adequate, even though a large sliding hiatus hernia exists.

There is little doubt however, that the presence of a sliding or combined hernia is associated with a higher incidence of oesophageal regurgitation than that in the normal population. The presence of the oesophago-gastric junction in the thorax therefore in some people interferes with the function of the cardiac mechanisms producing competence. This failure may be related to the altered pressure relationships about the oesophago-gastric junction, occurring as a result of the presence of this junction within the thorax. That this is not the only reason has been shown by Atkinson *et alii* (1958) in their withdrawal curves, where in some cases of hiatus hernia there is no gradient between the abdominal segment and the thoracic segment of the stomach. The tracings tend to show that the absence of the high pressure area in the lower oesophagus, corresponding to the position of the inferior oesophageal sphincter, is the most important factor in determining the presence or absence of regurgitation.

We cannot assume furthermore that in those patients with free reflux and a hiatus hernia, by replacing the stomach in the abdomen, by approximating the crura, or by increasing the oesophago-gastric angle we are restoring the normal mechanisms of competence. We are in all probability substi-

tuting secondary mechanisms to compensate for inefficient primary ones.

PATHOLOGICAL COMPLICATIONS

Of considerable interest to the surgeon are the pathological sequelae to the presence of the hiatus hernia and to the associated acid regurgitation.

The complications can be divided into two groups—

(a) Mechanical.

(b) Digestive.

1. *Mechanical*

The hernia of the hiatus is heir to the same complications as the inguinal hernia and of these irreducibility comes first. This may be due not only to secondary changes occurring in the oesophagus as a result of oesophagitis, but also to adhesion formation occurring between the sac wall and surrounding tissues, including the pericardium and pleura and to adhesion of the stomach within the sac. Thus even in large irreducible herniae good and permanent reduction can be obtained by dissecting free and adherent sac and stomach, for one may find in these an oesophagus which returns to normal length when the stomach is reduced below the diaphragm. Many irreducible herniae reach an enormous size but never give rise to any symptoms. These herniae must be mechanically stable, and the function of the stomach and the sphincters must be normal despite their abnormal positions. If they are mechanically unstable they will produce symptoms such as pain or dysphagia which arise because of a varying degree of distension of the stomach as a result of kinking, or because of an increase in the amount of stomach within the sac. These irreducible mechanically unstable herniae are dangerous because they may lead to obstruction and even gangrene of the stomach wall within the sac.

Case report

S.P. Male aged 81. This patient had symptoms of regurgitation for years. Barium meal in 1954 showed a moderate sliding hiatus hernia which was irreducible and visible on a plain X-ray of the chest.

Twelve months prior to admission he complained of increasing intermittent pain and dysphagia. Conservative measures produced some improvement, but finally he was admitted with acute oesophageal obstruction. Plain film of the chest revealed a considerable increase in the size of the hernia which now contained a fluid level (see Fig. IV).

At operation an incarcerated hernia was found with necrosis of portion of the stomach wall at the site of the hiatal margin. Reduction, repair of the hernia and oversewing of the stomach wall was performed.

FIG. IV. S.P., aged 81. X-ray of the chest showing a large hiatus hernia which contains a fluid level. Patient admitted in acute oesophageal obstruction and at operation incarceration of the stomach with obstruction was found.

As can be seen from this case and as will be emphasized later, these mechanical changes must be carefully sought for and it is wrong to be complacent when symptoms, which can be attributed to mechanical changes, subside on medical treatment; for medicines rarely solve mechanical problems.

Other complications can occur in the stomach in the hernial sac possibly because of interference with the venous drainage and/or stagnation in the herniated stomach. These complications, haemorrhage, superficial ulceration, deep ulceration and even perforation can be fatal.

2. *Digestive complications*

Better known are the complications arising in the oesophagus as a result of the action of acid gastric juice on the oesophageal mucosa. The commonest oesophageal change is reddening and oedema of the mucosa with a

tendency to bleed on contact, occasionally associated with shallow ulceration. The healing that follows shallow ulceration may be by the growth of gastric mucosa from the stomach at the expense of the normal lining. This leaves a state of affairs varying from occasional patches of gastric mucosa up to the so-called gastric mucosal lined oesophagus so well described by Barrett (1949). Deep ulceration may occur in the oesophagus as a result of the action of the regurgitant acid juice. In some cases this ulceration may occur in a portion lined by gastric mucosa (Barrett's ulcer), or it may occur at the junction of the two mucosae. Stricture is a common sequel to the development of deep ulceration occurring either at the gastro-oesophageal junction or at the site of previous ulceration in the lower oesophagus.

The simple explanation of the action of the acid gastric juice cannot be accepted without some demur, for many patients have oesophageal reflux with severe pain for many years but have very little to find on oesophagoscopy and never develop any recognizable ulceration nor a stricture. Familiar also is the patient who has been entirely asymptomatic until dysphagia brings him along for treatment of a well-established stricture. That some other yet unknown factor is required to act in conjunction with acid regurgitation, before significant ulceration will occur, seems a distinct possibility.

SYMPTOMATOLOGY

Much has been written concerning the symptomatology of hiatus hernia and of the difficulty which arises in connection with the differential diagnosis from gall-bladder disease and coronary artery disease. The good clinician today, with all the aids to diagnosis available, should rarely make a mistake in this regard.

The case histories of the last 150 in-patients discharged from St. Vincent's Hospital with a proven diagnosis of hiatus hernia, were analyzed. The average age, sex and principal symptoms and their duration were recorded (see Table 1).

As has been pointed out by many authors the disease occurs predominantly in females (73 per cent.) and occurs in the latter age group (average age 62). The common symptoms of epigastric and lower retrosternal pain with regurgitation and vomiting parallel other recorded series. Of great interest is the very high percentage in this series of haematemesis and melaena. It is not proposed to go into detail concerning all symptoms but to discuss only haemorrhage and dysphagia as the other symptomatic features of the disease have been covered by many authors.

TABLE 1

SYMPTOMS IN HIATUS HERNIA IN 150 CASES

Symptom	*Patients*	*Percentage*
Pain	122	81
Haemorrhage	65	43
Haematemesis		
Melaena		
Chronic anaemia		
Regurgitation	52	35
Vomiting	52	35
Flatulence	32	21
Dysphagia	24	16
Anorexia	16	11

Haemorrhage

As can be seen from Table 1, of the 150 patients discharged with the diagnosis of hiatus hernia 65 had had significant episodes of bleeding. It has been difficult in the past to see how massive haemorrhage can be related to hiatus hernia, so these figures were further examined, and the following facts were elicited. Of the 65 patients 15 (23 per cent.) had other gastric lesions, proven by barium meal or operation, which could have caused bleeding and these are excluded. In the remaining 50 patients 29 (58 per cent.) had radiographically demonstrable free oesophageal reflux, 13 (26 per cent.) had irreducible herniae, and 8 had neither irreducible herniae nor reflux reported on X-ray examination.

It is not unreasonable to suppose that haemorrhage comes either from the oesophagus or from the pouch of stomach in the hernial sac. If from the oesophagus, then acute erosions resulting from the action of the acid gastric juice are the most likely cause, while, if it arises in the stomach, then venous

congestion in an irreducible mechanically unstable gastric pouch is the most likely predisposing factor. That these may be the two factors responsible seems to be borne out by the figures, for 42 (84 per cent) of a total of 50 patients with hiatus hernia who had bleeding episodes, had either free reflux or an irreducible hernia.

The recognition of such an ulcer has important implications in treatment. It is commonly said that, if an ulcer is found during operation for hiatus hernia, simple reduction of the hernia will result in cure of the ulcer. This is true for ulcers occurring within the herniated gastric pouch; but those patients, who have a chronic ulcer on the lesser curve

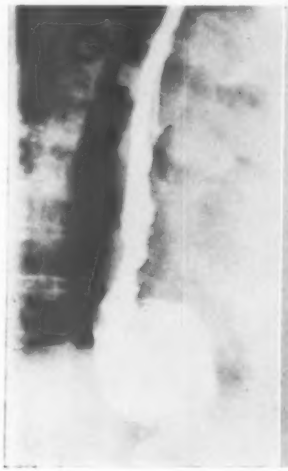

FIG. V. T.J., aged 85. Illustration of barium meal in 1955 when patient first complained of dysphagia.

FIG. VI. Illustration of barium meal of same patient one year later, showing stricture of lower end of oesophagus.

It cannot be denied with certainty that some of these patients may have had gastric erosions elsewhere in the stomach. These figures suggest, however, that a more realistic attitude should be taken to the patient who has a severe haemorrhage, and who has either a hernia with reflux or an irreducible hernia, and that operative repair should be more frequently recommended.

In this connection there is one important differential diagnosis which should be considered. The benign gastric ulcer high on the lesser curve or juxta-oesophageal ulcer is often a cause of diagnostic difficulty, especially when it is associated with a small hiatus hernia. This ulcer is difficult to demonstrate radiologically, or by any other means. It should always be considered in those people with small herniae without reflux who bleed, especially if the pain pattern or symptom complex do not exactly correspond to those for hiatus hernia.

not contained within the gastric pouch, must be treated by reduction of the hernia and gastric resection.

Dysphagia

This is an interesting and ill understood symptom in hiatus hernia. The normal function of swallowing demands the co-ordinated action of the muscular coats of a hollow tube, the lumen of which is unhindered by organic narrowing, occurring as a result of changes in the wall or resulting from pressure from without. Alteration of any one of these factors can occur in hiatus hernia giving rise to dysphagia.

Peptic oesophagitis in common with mucosal inflammation in other parts of the alimentary canal is associated with altered function of the underlying muscle coat, particularly that area underlying the segment of involved mucosa. This muscle spasm will occur with oesophagitis, which may be of a

mild degree, and will cause dysphagia. This type of dysphagia is a grave warning symptom, because an organic stricture can rapidly follow unhealed or persistent oesophagitis, and, should any trace of dysphagia persist following institution of treatment, repair is mandatory. Fig. V shows the barium meal in December, 1955, of an elderly man who complained of some retrosternal pain and dysphagia. It shows a small hernia with reflux and some oesophageal spasm. Despite adequate treatment, his symptoms persisted, and Fig. VI is a barium meal done one year later, when an organic stricture had developed.

Once an organic stricture has developed dysphagia becomes a constant and worrying symptom. Obturation of such a stricture can occur and result in acute oesophageal obstruction.

The final cause of dysphagia is pressure from a large para-oesophageal pouch. This is the mechanically unstable hernia which, by gastric distension or increase in the amount of stomach in the sac, produces oesophageal compression. In these herniae also dysphagia is a grave warning sign. It may be intermittent and spontaneously pass off, credit for which is incorrectly laid at the door of

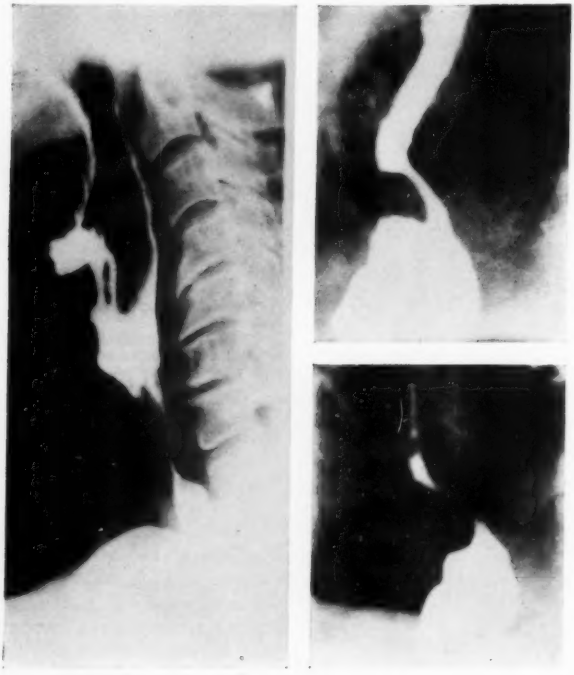

FIG. VII. Barium swallow of F.N., aged 46, showing hiatus hernia at lower end and spasm of cricopharyngeal sphincter at upper end.

The muscle spasm with oesophagitis does not always remain limited to the lower end and some patients will refer their dysphagia to the cricopharyngeal region where spasm of the sphincter can be demonstrated. In these patients the cause of the trouble is at the lower end. Fig. VII shows spasm at the upper end associated with a hiatus hernia.

medical treatment (see case report and Fig. IV).

Finally in discussing the association of dysphagia with hiatus hernia it is well to remember that the dysphagia may be due to other oesophageal disease. Oesophagoscopy is a very important diagnostic step in all

patients complaining of dysphagia, especially as such patients are most commonly in the older age groups.

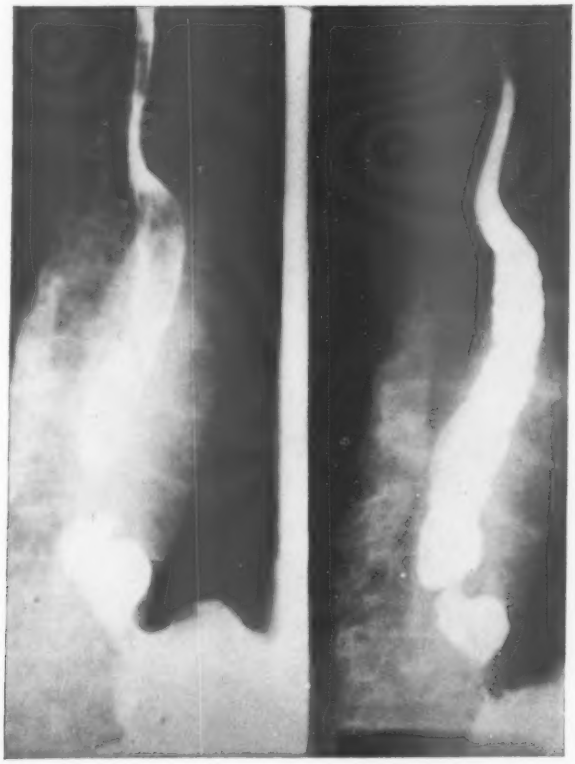

FIG. VIII. Barium swallow of Mrs. O.F., aged 60, showing hiatus hernia but no evidence of the carcinoma of the middle third of the oesophagus found on oesophagoscopy.

Fig. VIII is the barium swallow from a female patient aged 65 who complained of retrosternal pain with regurgitation for many months, but who for four months prior to admission to hospital complained of dysphagia. The X-ray shows a hiatus hernia but no oesophageal lesion. Oesophagoscopy, done as a routine in all such patients, revealed a carcinoma of the middle third of the oesophagus.

INDICATIONS FOR SURGERY

Medical treatment at present holds sway in the management of hiatus hernia, but it is important to point out that medicines have never solved mechanical problems, that the abnormality will in most instances be present for the lifetime of the patient and that it is associated with known severe complications. It is also equally important to realize that these complications frequently give very little warning and occur principally in the latter decades of life when surgical correction becomes the more hazardous.

The indications for operation can be classified under the headings of—

1. Failed medical treatment.
2. Haematemesis or other manifestations of bleeding.
3. Oesophageal complications.
4. Complications in the gastric pouch.
5. Associated disease.

1. *Failed medical treatment*

Most surgeons will agree that the small sliding hiatus hernia, which is difficult to

produce and the symptoms of which are all completely and permanently relieved by medical measures, does not require surgical repair. Weight reduction, as recently shown by Murphy (1958), plays a very important part in the medical programme. Should symptoms not be completely relieved after three months treatment, or should they persistently recur after periods of freedom, then the dangers of later complications are high and the hernia must be repaired. An important consideration influencing the decision to operate is the presence of irreducibility. If the hernia is difficult to demonstrate radiologically and obviously reducible, the tendency should be to conservative management. However, should an irreducible sliding hernia be present, even though symptoms may have been temporarily relieved, operation must be recommended for these are the patients who will almost certainly develop late complications.

The position of the para-oesophageal hernia is somewhat different from the last group. Many of these, although irreducible, are entirely symptomless and as long as they remain so can be safely observed. Once symptoms occur, however, the hernia is generally unstable. The disappearance of such symptoms may not be related at all to the medical measures which have been adopted, nor can it be regarded in any way as a guarantee of immunity to future severe and sudden complications. Once symptoms occur in the irreducible para-oesophageal hernia it should be repaired.

In the combined type of hernia, if the symptoms are considered to be entirely due to regurgitation and are completely relieved by symptomatic treatment, operation can be postponed with some degree of safety. Where symptoms are due to mechanical changes in the hernia, operation should be recommended.

2. *Haematemesis*

The association of bleeding with hiatus hernia may take the form of single or repeated episodes of haematemesis and melaena, of melaena alone, or of chronic iron deficiency anaemia. From the figures quoted earlier, the general principle should obtain that when significant haemorrhage has occurred in a patient who has a hiatus hernia demonstrated, operation should be recommended. This is particularly true if the hernia is associated with free oesophageal reflux or is irreducible.

3. *Oesophageal complications*

Once significant oesophagitis or any of the more serious complications has occurred, operation becomes essential; the only contraindications being extreme age or associated severe disease. No unanimity of opinion exists on the ideal method of treatment once oesophageal stricturing has occurred. It is our opinion that the best results are obtained by simple reduction of the hernia and dilatation of the stricture, when the hernia can be completely reduced. Complete freeing of the oesophagus in these cases makes reduction easier and often enhances, to a surprising degree, the ease of dilatation of the stricture. Where complete reduction is impossible and oesophageal changes do not allow ready dilatation, proximal oesophago-gastrectomy with oesophago-gastrostomy, pylorotomy and vagotomy is the best method of dealing with the situation.

4. *Gastric complications*

These may occur within the herniated gastric pouch, when they constitute an absolute indication for operation. Gastric and duodenal ulceration, in association with hiatus hernia, is not uncommon. Nineteen cases of the 150 had proven gastric or duodenal ulceration, excluding those with ulceration in the gastric pouch. The presence of this additional lesion constitutes an added indication for surgery.

5. *Associated disease*

The presence of cholelithiasis constitutes an additional indication for operation and in nine cases out of the total series this association existed. With regard to the question of associated disease, it is interesting to point out that in none of the 58 cases operated upon (34 by the abdominal route) was any unsuspected intra-abdominal disease found.

SURGICAL TECHNIQUE

Repair of a hiatus hernia can be performed by an abdominal or a thoracic approach. The thoracic surgeon feels that the approach from above is preferable, because of the ease of exposure. The abdominal surgeon, because of the fairly high incidence of other abdominal pathology, feels that the abdominal route is preferable. Such is the nature of things that neither will be able to convert the other completely to his way of thinking. However, both can agree that it is only by very careful attention to anatomical detail

Both must also recognize that certain circumstances make one approach or the other mandatory. The absolute indications for the abdominal approach are:

(a) Presence of associated intra-abdominal lesions such as cholelithiasis, duodenal ulceration.

(b) Presence of left sided chest disease, pleural thickening from an old empyema, basal tuberculosis.

(c) Marked emphysema or other pulmonary disease.

The absolute indications for the thoracic approach are:

(a) Presence of permanent oesophageal complications.

(b) Large irreducible para-oesophageal or sliding herniae.

The operation is performed under intratracheal anaesthesia, with relaxants and the patient in the lateral position with the left side uppermost. A small rubber cushion under the right lower chest helps exposure, and, with the table rolled forward slightly, the liver and intra-abdominal viscera tend to fall away when the diaphragm is opened.

Skin infiltration with 1/200,000 adrenalin is used to cut down bleeding from the incision. This is along the line of the eighth rib from the costal cartilage in front to a point three fingers breadth from the spine posteriorly. The muscles are divided in the line of the rib which is subperiostially removed; the pleura opened and a chest wall retractor inserted. The inferior pulmonary ligament is divided and the lower lobe retracted upwards. The pleura is opened longitudinally along the line of the oesophagus to the free margin of the hiatus, where the incision is carried at right angles backwards and forwards along the margin of the hiatus. Stay sutures are placed in each free edge of the divided pleura, which is reflected off the oesophagus and hernial sac. The hernial sac, containing the stomach, can then be easily seen, and the height of the gastro-oesophageal junction above the hiatus determined (see Fig. IX). In many of the large herniae, especially the irreducible ones, adhesions between the sac and pericardium and between oesophagus and mediastinal tissues can be well seen. The oesophagus is then dissected free from its bed and a tape passed around it.

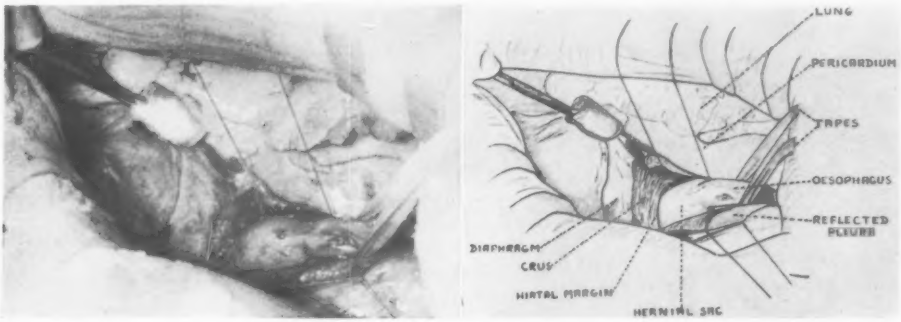

FIG. IX. Photograph and diagram at operation showing oesophagus, oesophago-gastric junction, hernial sac and hiatal margin.

These mainly apply, because it is often impossible to secure complete freeing of the oesophagus or sac from below, and, where resection of oesophagus or proximal stomach is required, adequate exposure is essential.

There are two groups of patients where some argument is possible. The first group comprise those in whom some other intra-abdominal disease is suspected, but not proven and in these the abdominal approach is preferable. The second group are those patients with a moderate sized irreducible hernia. For the reasons given earlier, these are better approached from above.

Thoracic approach

The technique of repair from above is illustrated in a series of illustrations and photographs taken during an actual operation.

A radial incision in the diaphragm is then made commencing over the central tendon

and in the line of the muscle fibres. A small incision is first made and then the under surface of the diaphragm is carefully inspected to see the course of main phrenic vessels and nerves. The diaphragmatic incision is then carried back towards the hiatus to within 4-5 cms. of the margin. The final incision is 8-9 cms. in length, and care is taken to avoid the major divisions of the phrenic nerve (see Fig. XIII). It is possible to carry out a laparotomy through such an incision and the stomach and gall-bladder should be carefully examined. The edges of the diaphragmatic incision are held retracted by stay sutures.

its attachments to pericardium and mediastinal tissues and the margin of the hiatus cleared. With the abdominal hand putting tension on the sac wall, the outer layer or phreno-oesophageal ligament is divided 2 cm. from the attachment to the oesophagus. This is shown in Fig. XI, where a suture has been placed through the divided ligament for demonstration. The extraperitoneal tissues and peritoneum are revealed following division of the ligament. At operation the incision is carried through all layers of the sac wall and, once a short segment of the ligament and peritoneum have been thus ob-

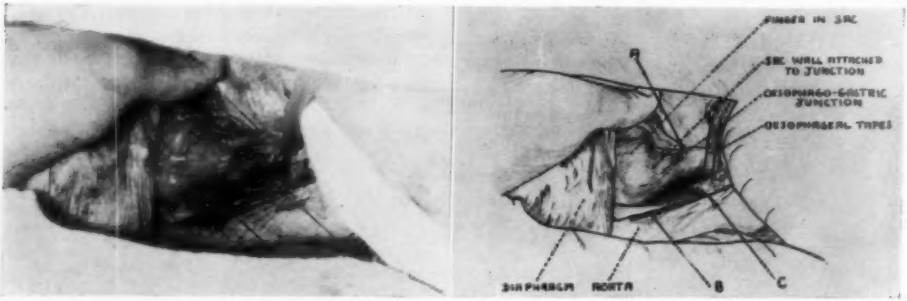

FIG. X. Illustration showing index finger in hernial sac and demonstrating attachment of sac wall to oesophago-gastric junction.

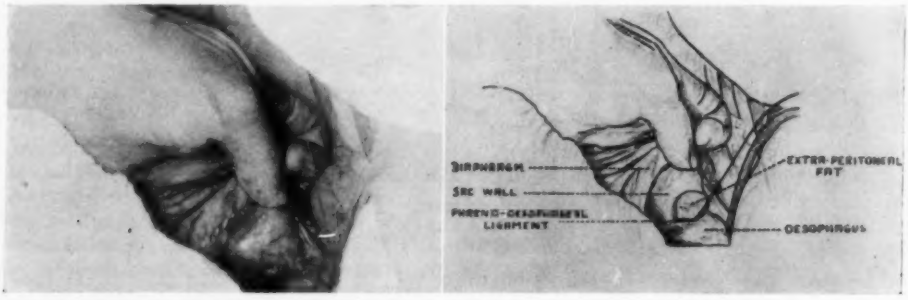

FIG. XI. Illustration of the division of the phreno-oesophageal ligament exposing the extraperitoneal fat. A suture has been placed through the oesophageal end of the ligament for demonstration purposes.

The index and second finger are next passed up into the sac from below and the size of the sac, the size of the hiatus, and the site of attachment of the sac wall are determined. The sac lies anteriorly and laterally and the extent of the attachment of the sac wall, comprising peritoneum and phreno-oesophageal ligament, to the gastro-oesophageal junction is carefully defined (see Fig. X). The sac is next freed completely from

tained, a suture is doubly passed through all layers and held. The dissection then proceeds with further division of phreno-oesophageal ligament and peritoneum, with insertion through them of a series of sutures, so that finally the whole length of the attachment of the ligament, together with the peritoneum, is held by 7-8 sutures (see Fig. XII). In this diagram, a close-up view, sutures A and B have been passed, for demonstration,

through the ligament and peritoneum separately. The careful dissection of the attachments at the gastro-oesophageal junction and their preservation are the most important parts of the procedure. If the sac wall is carelessly detached and the hernia reduced by traction on the stomach before the sutures have been placed, the peritoneum and ligament are separated and the oesophageal end of the ligament often lost as a result of overstretching of the fascia propria. It is important that the sutures, which will keep the hernia reduced, should be placed before the hernia is reduced. These sutures are then passed down through the hiatus in order and by traction on them the hernia is easily reduced (Fig. XIII). Where much perioesophagitis is present, further clearing of the oesophagus up to the level of the inferior pulmonary vein or higher, may be necessary. The sutures are then inserted through the under surface of the diaphragm, 2 cm. from the free margin, taking a good bite of the diaphragmatic muscle.

By holding up the oesophageal tape it is possible to identify by palpation the two limbs of the crus (Fig. XIV). These are carefully cleared, and two sutures passed through them. A good bite of the right limb is essential and can best be obtained by mounting the needle in the needleholder in reverse fashion and inserting the stitch from above down. The crura are approximated behind the oesophagus by two or three sutures (see Fig. XV).

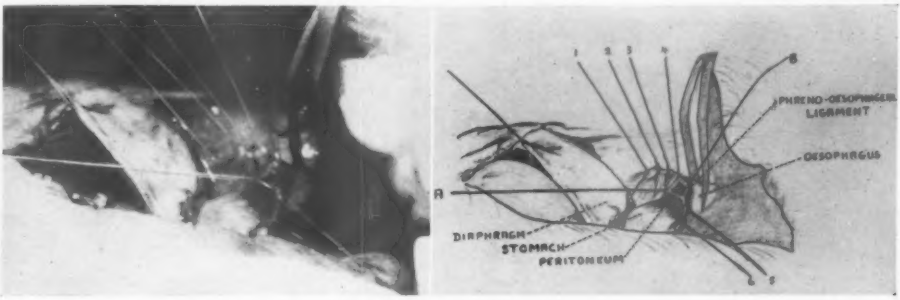

FIG. XII. Highly magnified view of series of sutures through the phreno-oesophageal ligament and peritoneum. 1—6 inc. Sutures A and B have been passed through the peritoneum and phreno-oesophageal ligament separately for demonstration purposes.

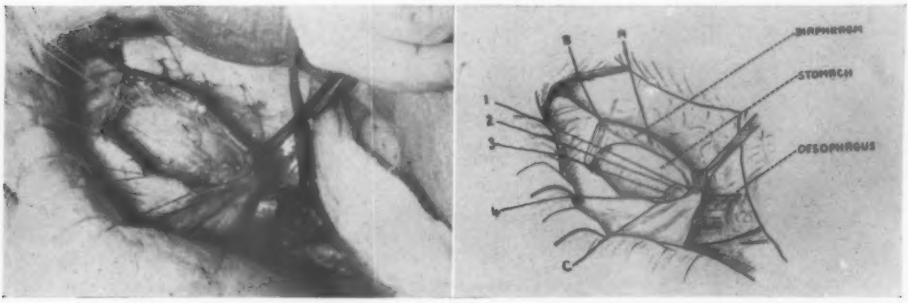

FIG. XIII. This shows how the sutures have been passed down through the hiatus and how traction on them reduces the hernia. A, B, C are stay sutures in cut edges of diaphragm.

A Hurst mercury bougie is then passed and it should be possible to insert the tip of the finger through the hiatus alongside the bougie.

The oesophageal tape is removed, and the pleura loosely closed over the oesophagus, leaving an aperture at the inferior end for drainage. The diaphragm is closed by a continuous thread suture, and the chest cavity

drained by an intercostal tube through the tenth space for twenty-four to thirty-six hours.

It is important that the vagus nerves be carefully preserved during the procedure and not handled carelessly as this may worsen the post-operative gastric distension which sometimes occurs. If vomiting occurs, a Rehfuss tube is passed and emptying the stomach is usually sufficient to prevent further trouble.

such is the case and division of the offending suture offers the quickest and surest method of relief.

The operation from below

The patient is placed supine with the head of the table slightly elevated. An upper midline incision is used. Excision of the xiphisternum greatly increases the exposure of the operative field.

FIG. XIV. This figure illustrates the exposure of the crural limbs behind by drawing up the oesophagus.

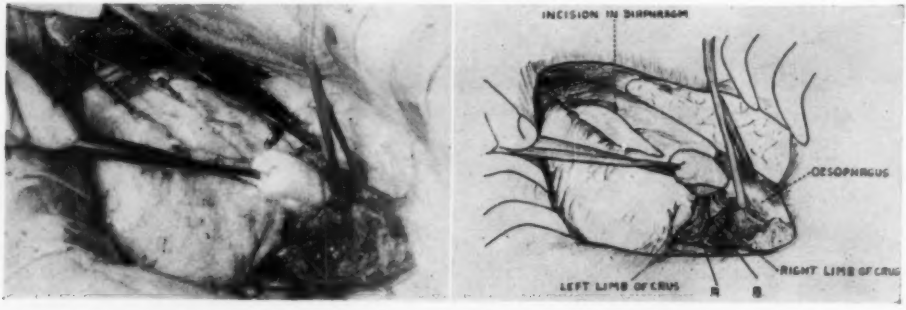

FIG. XV. The crural limbs have been approximated by two sutures A and B behind the oesophagus.

An X-ray of the chest on the first post-operative day will exclude any significant haemothorax or pneumothorax. On the third to the sixth day it is frequent for these patients to complain of temporary dysphagia due to oedema about the operation site. This passes off in a few days, and is an indication that the suturing is correct. If the dysphagia persists into the third and fourth weeks, one must suspect too tight apposition of the crura. Should dysphagia for solid food be present after two months, one can be certain that

After a thorough abdominal exploration to exclude disease particularly in the stomach and duodenum, gall-bladder, pancreas and colon, the left triangular ligament is divided and the left lobe of the liver retracted well to the right. Then, by traction downwards on the body of the stomach, the hernia is reduced below the diaphragm (Fig. XVI, A and B). The anterior free wall of the hernia, consisting of peritoneum and underlying phreno-oesophageal ligament, now lies directly in front of the abdominal oesophagus.

The next step in the operation is the exposure and further mobilization of the oesophagus and to do this the overlying peritoneum must be divided. It is most important, at this stage, to preserve the phreno-oesophageal ligament as a definite structure by dividing the peritoneum, and with it the underlying ligament, quite close to the diaphragm, as is shown in likely to be missed altogether. We believe this to be a major mistake.

At all events the abdominal position of the oesophagus is exposed and then freed around its whole circumference by gentle blunt dissection with the fingers. Posteriorly, if the lesser sac is high, it may be necessary to

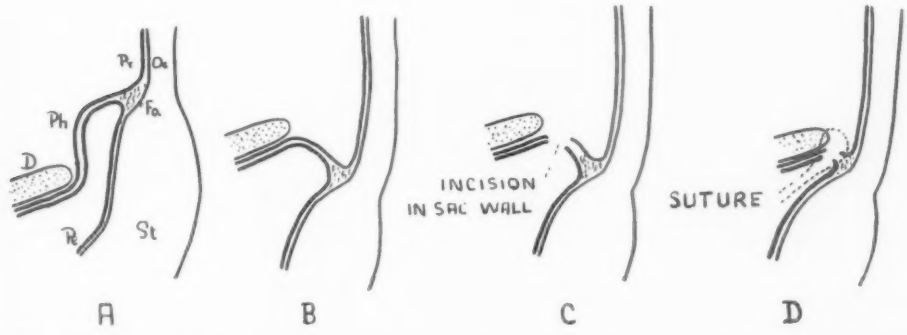

FIG. XVI. Illustrating some essential steps in the operation from below. A—The hernia unreduced. B—The hernia reduced. C—The site of election for division of the sac wall. D—After excess sac wall has been excised the suture shown is so placed to reconstitute the phreno-oesophageal ligament and the peritoneum.

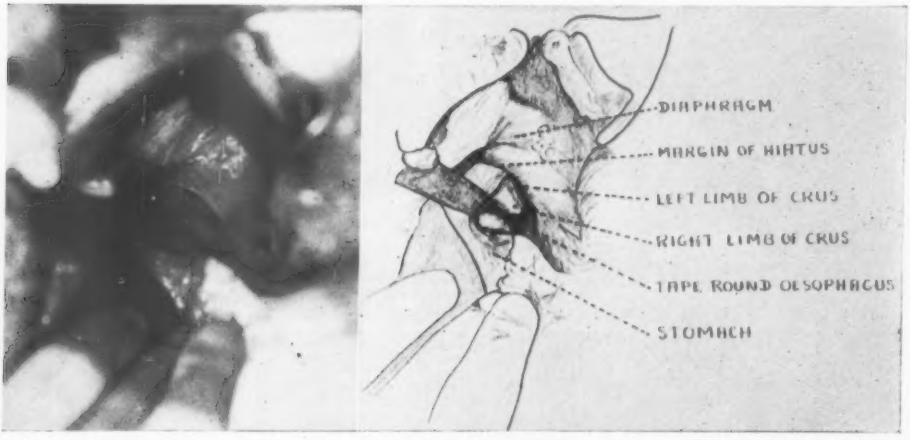

FIG. XVII This is a view of the oesophageal hiatus after the oesophagus has been mobilized and retracted to the left. Note the two thick muscular bellies of the crural limbs and the sharp anterior margin of the hiatus.

Fig. XVI C). The two layers are applied quite closely to each other over the major part of the sac wall, but near the cardia they veer away from each other and are separated by a good deal of loose areolar tissue. If the peritoneum is divided too close to the cardia the phreno-oesophageal ligament is divide a further layer of peritoneum. The oesophagus is then mobilized as much as necessary and retracted well forwards and to the left (or right), to expose the two limbs of the right crus of the diaphragm (Fig. XVII). By careful dissection, at this stage, it is possible to expose the broad muscular bellies of

the right crus and to trace the two limbs upwards as they diverge to surround the oesophageal hiatus. Anteriorly they, of course, form the sharp anterior edge of the hiatus. It is easy to see now that the oesophageal hiatus is not a vertical opening, but one which slopes in a postero-inferior direction from front to back. The anterior border is sharp and knife-like. The posterior border is a broad structure, consisting of the muscular bellies of the crural limbs.

The first stage in the repair is the narrowing of the hiatus by the approximation of the two crural limbs. This is accomplished by first of all retracting the oesophagus well to the left. The left limb of the crus is then grasped by a long Babcock or volsellum forceps and brought forward and to the right, delivering it into the centre of the operative field. A mercury bougie is then passed down the oesophagus by the anaesthetist. The two crural limbs are then sewn together by two or

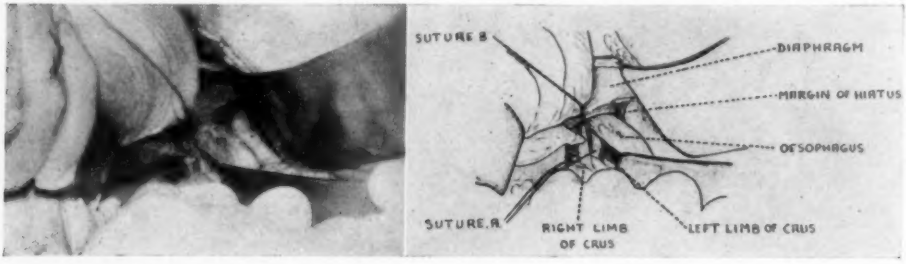

FIG. XVIII. This is a photograph taken during the repair of the hiatal orifice. The two limbs of the right crus are being sutured together behind the oesophagus, which is drawn well to the right.

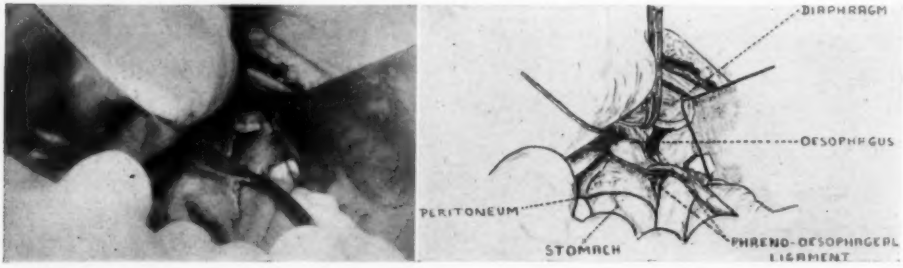

FIG. XIX. The stage is set for the anterior part of the repair. The excess peritoneum and phreno-oesophageal ligament has been excised and the two ends of the latter structure are held in long artery forceps ready for the insertion of the anterior sutures.

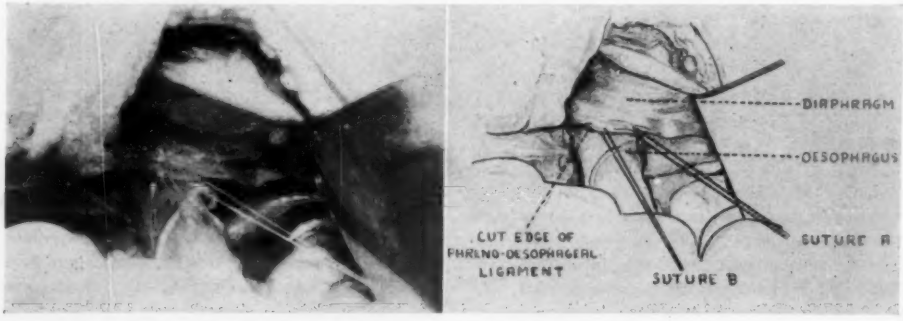

FIG. XX. The anterior sutures are first placed superiorly. The first two of these are shown in place, passing through the peritoneum, the phreno-oesophageal ligament and the sharp margin of the diaphragm.

three deep linen sutures. The hiatal orifice is narrowed sufficiently to permit only the tip of the index finger to fit between the diaphragm and the oesophagus (Fig. XVIII). The oesophagus is then laid back in its bed and the repair continued anteriorly.

The anterior repair consists of the excision of the redundant peritoneum and phreno-oesophageal ligament and then their reconstitution by stitching. This is done by suturing the free margin of these two structures at the diaphragm to their free margin at the cardio-oesophageal junction (Fig. XIX). It is easier to place the sutures through the upper margin first (Fig. XX, sutures A and B). Usually three or four sutures are necessary. through the cut edges of the phreno-oesophageal ligament and peritoneum below (Fig. XXI). After insertion, the sutures are tied and the operation is completed.

It has been the purpose of this paper to illustrate some points in connection with the anatomy, physiology and complications of hiatus hernia.

The important association of haemorrhage with hiatus hernia has been emphasized and a plea for a more realistic attitude to this problem has been made.

The surgery from the thoracic and abdominal approaches has been illustrated and the importance of the recognition of the anatomy involved in repair emphasized.

An attempt to equilibrate the widely differing surgical attitudes to the problem has been made. That there are absolute indications for the surgical approach to this problem from above and below has been pointed out, but it must be agreed finally that in many other cases the surgeon's particular point of view will determine his surgical approach.

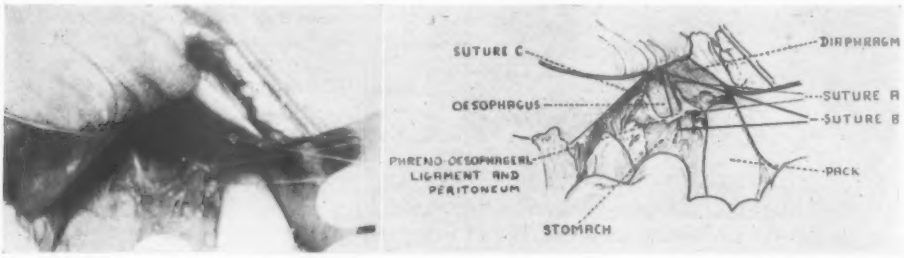

FIG. XXI. This shows the anterior sutures placed through the lower margin of the phreno-eosophageal ligament and peritoneum at the cardia. When these sutures are tied and the two margins approximated the anterior repair is completed..

CONCLUSIONS

It is hoped that in this discussion on hiatus hernia sufficient emphasis has been placed on the need for surgical repair in the great majority of patients. The failure to recognize the warning signs in the mildly persisting symptoms of the patient on medical treatment will result in an increasing number of severe complications, such as oesophageal stricture, in the latter years of the patient's life.

Follow-up of medically treated patients must of necessity be an extremely long one, for the complications which early surgical repair could avoid may not occur for as long as twenty years after recognition of the condition. It is important to realize that relief of symptoms by medical measures is not the cure of the disease.

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EXPERIMENTALLY INDUCED TUMOURS OF THE INTESTINE

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THE improvement in the results of management of tumours during this century is apparent to all who are able to compare the results of modern therapy with those of thirty to forty years ago. This is partly due to changes in surgical treatment and modifications in the application of deep X-ray therapy and is largely shown in the disappearance of infections, obstruction to tubes and other complications. The large fungating, ulcerating growths of past decades (a not uncommon sight in the wards of a hospital of those days) and other distressing complications have now almost vanished. The improvements, therefore, are due to a tremendous reduction in morbidity.

It is apparent, however, as in other fields, that the only really satisfactory answer to the problem of the malignant neoplasm is prevention. This necessarily implies and demands a thorough knowledge of the basic features of carcinogenesis. Fundamental cancer research has been proceeding at a rapid rate in recent years and advances have been made possible mainly by animal experiments. Furthermore, this has been achieved because of the recently increased knowledge of the carcinogens, specifically the chemical carcinogens.

Naturally-occurring neoplasms in animals have been valuable both because of the possibility of observing all stages of the disease and also the practicability of studying the effect of the many factors which may influence the development and progression of the growths. The study of the chemical carcinogens has, of course, made possible the more precise and minute investigations of tumours of all stages and also has provided numerous examples in circumstances and under conditions specially favourable for their study.

Although tumours of many organs either occur naturally or may be produced readily, naturally-occurring bowel neoplasms in animals are extremely uncommon. Furthermore, considerable difficulty has been experienced in producing tumours here and only relatively recently have consistent results been obtained.

REVIEW OF PREVIOUS WORK

The observation that naturally-occurring tumours of the bowel in animals are rare is all the more significant when it is found to apply to the commonly used experimental animals which may live throughout their normal span without being subject to the hazards of food deprivation, predators or the like. Accounts of the somewhat meagre material obtained from laboratory animals have been recorded since the beginning of the century.

Neoplasms of the small intestine, as in man, are much rarer than those of the colon. Occasional examples have been described in mice by Bashford, Murray and Cramer (1905), Bashford (1906), Murray (1908) and Heidenhain (1929) amongst others and in the rat by Bullock and Curtis (1930) and Curtis, Bullock and Dunning (1931). The general rarity of bowel tumours was indicated by the demonstration of 19 examples of carcinoma of the bowel in 142,000 mouse autopsies (Wells, Slye and Holmes, 1939). Thus, for satisfactory work in the laboratory, experimentally produced lesions were clearly necessary.

Bowel tumours have been induced successfully in several ways, which fall naturally into two main groups:

(i) Oral administration of a single large dose or repeated doses of the isotope, yttrium^{91} (by stomach tube) produced 4 bowel tumours in 33 rats and 6 tumours in 8

rats respectively (Lisco, Brues, Finkel and Grundhauser, 1947). General irradiation by X-rays (Brecher, Cronkite and Peers, 1953) and by fast neutrons (Nowell, Cole and Ellis, 1956) also produced these tumours; this may be related to the susceptibility of bowel mucosa to irradiation injury. It indicates that a powerful source of irradiation does not necessarily need to be located in the bowel itself to produce a local effect there.

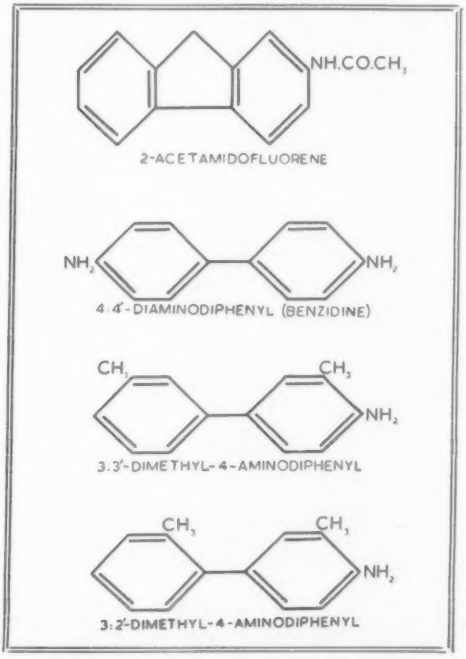
(ii) The effective chemical carcinogens have fallen into two chemical types: the "carcinogenic" hydrocarbons and the aromatic amines.

The condensed polycyclic hydrocarbons, since their production in pure form nearly three decades ago, have been used principally on the skin but more recently have been applied to other regions including the bowel. Administration of 1:2-5:6-Dibenzanthracene and 20-Methylcholanthrene by mouth, in various media, has produced tumours of the bowel (Lorenz and Stewart, 1940; Stewart and Lorenz, 1946; Stewart, 1953); on the other hand such application may be ineffective (Laurens and Bacon, 1952); incidentally, oral administration of lubricating oil has also been followed by tumour formation (Lushbaugh and Hackett, 1948). Local application and injection of 20-Methylcholanthrene into the wall of the colon have produced an occasional tumour (Rack, Kaufman and Simeone, 1955) as has rectal injection of alcohol (Krebs, 1928). It is noteworthy that bowel tumours followed the introduction of 20-Methylcholanthrene subcutaneously (Miller and Pybus, 1955).

Attention was directed early to the aromatic amines because their relation to bladder cancer was demonstrated in man. During studies of some of these substances it was found that 2-Acetamidofluorene produced some intestinal tumours (Cox, 1937; Bielschowsky, 1944; Dunn and Kessel, 1945; Symeonides, 1954; Morris, 1955). The demonstration of the carcinogenicity of benzidine (4:4'-Diaminodiphenyl) in rats, with production of acoustic, gland, liver and bowel tumours (Spitz, Maguigan and Dobriner, 1950) not only revolutionized various activities including the well-established benzidine test but showed the importance of other members of this general chemical group. Growths were also produced using NN-Dimethyl-4-aminodiphenyl (Miller, Miller, Sandin and Brown, 1947).

TABLE 1

Formulae of aromatic amines which, amongst others, have been used as carcinogens in experimental animals.



Following these observations other diphenyls have been studied. Walpole, Williams and Roberts (1952, 1955) employed the compound 3:2'-dimethyl-4-aminodiphenyl and produced a large number of intestinal neoplasms in Wistar strain rats. Several related compounds have also been effective (Walpole and Williams, 1958). This is clearly the most valuable method to date for this purpose and advantage has been taken of it here to attempt to determine to what extent they resemble or mimic the naturally occurring neoplasms of man.

It is not intended, nor is it appropriate, here to discuss the interesting work which has been carried out on the numerous compounds obtainable nor the light that this is

throwing on the molecular site of action of these materials. This has been discussed recently at length by Haddow (1958) and Walpole and Williams (1958) amongst others and reference may be made to these papers for special chemical details.

FIG. I. Colour photograph showing multiple tumours in the sigmoid colon and rectum of a rat injected with 3:2'-Dimethyl-4-aminodiphenyl; specimen obtained on 287th day. Bowel has been opened to demonstrate the nodules from the mucous aspect.

ANIMAL EXPERIMENTS

The animals were random mated Wistar strain rats which had been maintained as a closed colony in the laboratory at the University of Melbourne for several years. Two groups were taken (A) rats 3½ months old weighing from 100 to 140 g. and (B) others 6 months old weighing 150 to 250 g. They were kept in galvanized iron cages and fed liberally on a routine standard diet (Barastoc pellets — a local proprietary food) with free access to tap water.

Twelve males and 12 females from the first group were given 3:3'-Dimethyl-4-aminodiphenyl and 9 males and 9 females from the second group 3:2'-Dimethyl-4-aminodiphenyl by subcutaneous injection in the abdominal wall, alternately on each side. The compounds were dissolved in arachis oil, 4 g. in 100 mls., and administered in daily doses (5 days a week) of 0.5 mls/ 100 g. body weight. Controls were given similar amounts of simple arachis oil. The animals failed to gain weight at the same rate as the controls and, after six months, sometimes lost weight; at these times the injections were discontinued for a short time. At the end of the experiment, surviving animals weighed from 150 to 340 g. (in Group A) and 250 to 320 g. (in Group B).

The time of the experiment was just less than one year; the shortest time any animal was observed was 145 and the longest 351 days. The total amount of chemical given to an animal ranged from 240 to 500 mg.

TABLE 2

The number and distribution of tumours (in the case of multiple tumours only the largest growth is included) in the two groups of animals in the experiment. Group A contained rats injected with 3:3'-Dimethyl-4-aminodiphenyl and Group B with 3:2'-Dimethyl-4-aminodiphenyl.

	No. of Animals	*Salivary Gland*	*Stomach*	*Small Intestine*	*Colon*	*Rectum*	*Liver (Sarcoma)*	*Mammary Gland*	*Uterus*	*Thigh (Rhabdomyoma)*	*No. of Tumours*
Group A											
Males	12	1	1	2	2	–	1	–	–	–	7
Females	12	4	–	1	–	–	–	1	1	–	7
Group B											
Males	9	–	–	–	3	2	–	–	–	1	6
Females	9	–	–	–	4	3	–	–	–	–	7
Total	42	5	1	3	9	5	1	1	1	1	27

RESULTS

In the 42 animals, 27 showed one or more malignant tumours. These all appeared before the end of a year from the beginning of the experiment. Five animals died during the early part of the period and these showed no tumour; the cause of death in two was not determined with certainty but in the other three it was due to a streptothrix pneumonia occurring in epidemic form (the organism being a common and usually saprophytic inhabitant of the upper respiratory tract and mouth). Four of the control animals also died of pneumonia of this type. Two other animals were found to have nematode infestation of the liver and, in one, peritonitis had developed following its entry into the peritoneum.

FIG. II. Colour photograph of the abdominal contents of a rat seen when the cavity was first opened. The descending colon has been opened to show a fungating tumour. Other growths are apparent from the peritoneal surface of the colon; there is local gross distension.

When animals appeared to be deteriorating or if they developed signs suggesting the presence of a tumour, such as passage of blood per rectum, they were killed and investigated. Results of study of those dying naturally were known to be unsatisfactory owing to rapid tissue autolysis occurring in small animals. In some cases no tumour was found. In order to obtain early lesions (in the precancerous phase), all surviving animals were killed and examined on the 351st day of the experiment. It seems definite,

FIG. III. Colour photograph showing several tumours in the colon and rectum. This has been opened, in part, to demonstrate them from the cavity; others were present in the unopened part of the gut.

FIG. IV. Colour photograph showing tumours of the intestine, with an ovarian cystic tumour in a rat. The histological appearances of the ovarian growth showed it to be secondary to the adenocarcinoma of the bowel.

therefore, that the number of cases of fully-developed tumour can be regarded as a minimum yield; more would be expected to have arisen at a later date, as has been observed in other experiments (Walpole, Williams and Roberts, 1952).

Tumours occurred at various sites which are given in Table 2. The total number of tumours was almost the same in each of the two groups, though there were fewer animals in Group B; on the other hand, these animals were somewhat older at the beginning of injections and, in view of the known influence of age on tumour development, might have been expected to exhibit their growths earlier. No tumour was found in control rats during the period of the experiment nor in a few animals kept for 600 days.

thigh) and it was assumed, for histological reasons, that these were independent neoplasms.

In three cases the predominant growth was in the small intestine at distances ranging from 3 to 30 cm. from the ileo-caecal valve; they were found at 259, 345 and 351 days. Of the 14 large bowel tumours, three occurred in the caecum (found at 282, 296 and 340 days), 5 in the rectum (found at 271, 273, 274, 287 and 351 days) and the remainder in various parts of the colon (found at 253 269, 280, 285, 295 and 350 days). In that two-thirds of the cases showed multiple tumours and only one is referred to in each case, it is clear that more than 17 growths were found at the various sites. Since some

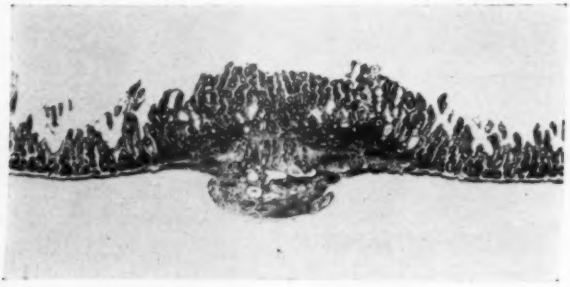


FIG. V. Photomicrograph of a small early carcinoma of the small intestine of the rat, showing infiltration through the wall to involve the peritoneum. (x 12)

Intestinal tumours comprised 17 examples (not including a gastric carcinoma); though other growths were less numerous (see Table 2), several of them arose at relatively early stages of the experiment (for example, rhabdomyoma of the thigh at 145 days and carcinoma of the breast at 272 days and therefore diminished the chance of localization of tumours in the bowel. Salivary tumours, as noted in other series, were relatively common (5 cases).

Twelve of the 17 animals with intestinal tumours showed multiple growths (Fig. I). In all cases the number of examples cited refers to the number of animals; only the largest growth in a given case is referred to in the Table. In two cases a second growth occurred outside the bowel (mediastinum, nodules were of uncertain nature and their classification thus a matter of opinion, a precise statement of numbers is not given.

The growths presented in various forms. The fungating tumour was the most frequent and occurred in all parts of the gut (Figs. II and III); its frequency is doubtless to be correlated with the frequence of intussusception. Sessile nodules occurred often and occasionally extension along the wall gave the typical "garden-hose" appearance. Stricture was uncommon but was seen in three cases. Macroscopic ulceration, even in cases presenting with rectal haemorrhage, was uncommon. All of these growths could be recognized on external examination of the bowel, either as enlargements, thickenings or infiltrations.

The remarkable feature of these tumours was their multiplicity; as many as five well-defined growths occurred and sometimes there were very numerous small nodules in one specimen. This is to be distinguished from the numerous multifocal areas of proliferation observed histologically. The tumours occurred irregularly; sometimes they were in ileum and rectum and at other times in various parts of the colon. Often irregular thickenings were found throughout the bowel, the mucosal increase being even more obvious microscopically than it was on examination of the gross specimen.

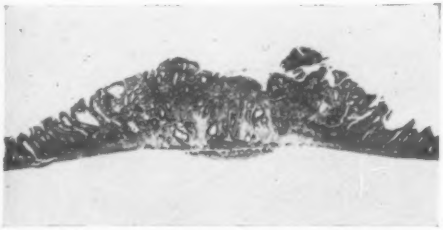


FIG. VI. Photomicrograph showing a small bowel tumour in which there is involvement of the mucous membrane with early ulceration (Cf. Fig. V). (x 8)

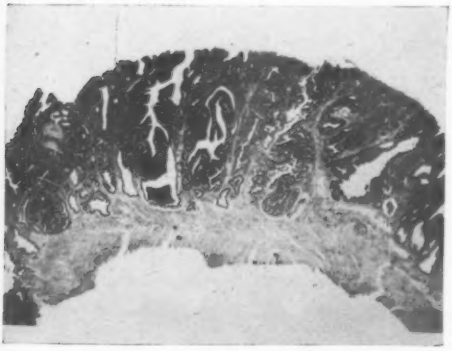


FIG. VII. Photomicrograph of rat colon showing part of an extensive carcinoma; the irregularity of the epithelium and the invasion of the muscle wall is shown. (x 8)

Metastases, in the peritoneal cavity, were observed in 3 cases only: two on the serosal surface at a distance from the primary growth and one in the ovary (Fig. IV). In one of these there was a peritoneal accumulation of mucus such as is seen in pseudomyxoma peritonei. Tumours in other regions, such as the mediastinum and thigh, were regarded as being different from the intestinal tumour, although it is recognized that histological appearances may be misleading as regards histogenesis. Metastatic tumours were not found in lungs or liver.

Intussusception occurred in 5 cases and intestinal obstruction from an occluding and stricturing carcinoma of the colon was observed in one.

Histological examination

Study of frozen sections of fresh tissue was carried out on almost all tumours and paraffin sections of fixed material were made in all cases. Various fixatives (Flemming, Carnoy, Bouin) were used (on different pieces of tissue) in some cases but 10 per cent. formol-saline was employed routinely. Haematoxylin and eosin stain was regularly employed, special stains such as Masson's trichrome, Van Geison or Alcian Blue being used only occasionally.

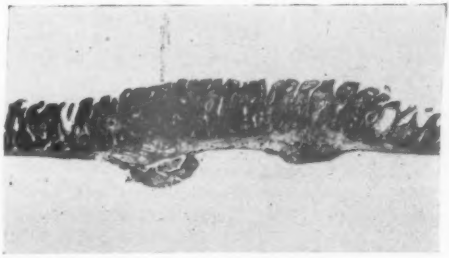


FIG. VIII. Photomicrograph of portion of bowel wall showing two foci of neoplastic change with invasion of the wall and serosal involvement. (x 7)

The growths showed a variable degree of activity. In some, though there was only slight and localized proliferation, invasion of the intestinal wall indicated their nature (Fig. V); in others the degree of change was gross. The glands in the mucosa were enlarged and often cystic (Fig. VI), the cells lining them being larger and staining differently from their normal counterparts. Various epithelial changes, which correspond with those described in other glandular neoplasms, need not be discussed here in detail. Infection of the superficial layers, with ulceration, was apparent in several of the larger growths.

Apart from the cases where considerable lengths of bowel wall were involved in the neoplastic change (Fig. VII), other examples showed separate foci (Fig. VIII), sometimes numerous, demonstrated by an invasion of the muscular layers of the wall; these established explicity, if such were necessary, the multifocal distribution of activity of the carcinogens.

FIG. IX. Photomicrograph of glands invading muscle and showing numerous mitotic figures. (x 380)

The number of cells in the glands was increased so that the glands often became replaced or represented by solid cords of cells; the cells varied considerably in size and form. Nuclei were variable in form and prominent; particularly in the early stages of development mitotic figures were numerous (Fig. IX). In general, goblet cells were diminished in number but mucin was occasionally conspicuous.

In the neighbourhood of the proliferating epithelium there was a hyperplasia of muscle cells of the muscularis mucosae and the inner circular muscle as well as the connective tissue; cells enlarged and developed prominent nuclei (Fig. X). Contraction of connective tissue and muscle resulted in invagination of the affected zone into the lumen of the bowel so that a polypoid structure was formed (Fig. XI). The features of many of these are characteristic of malignancy despite the absence of peritoneal involvement.

Most of the growths were of typical adenocarcinomatous type. Where infiltration by a tumour occurred, the neoplastic tissue often adopted an indifferent spheroidal cell form.

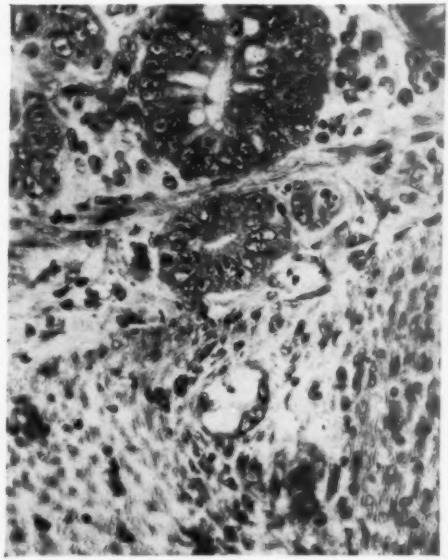


FIG. X. Photomicrograph of a section of rat intestine showing smooth muscle and connective tissue hyperplasia near glandular downgrowths. (x 380)

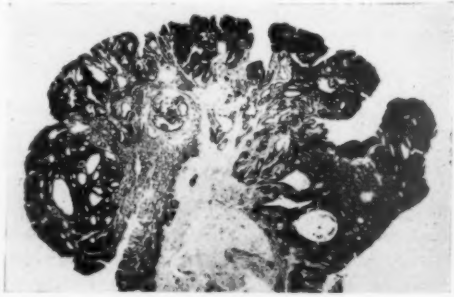


FIG. XI. Photomicrograph of a polyp of the colon showing beginning invasion of the stalk by atypical glands. (x 8)

Squamous metaplasia was seen in one rectal carcinoma. A mixture of cell types was sometimes found (Fig. XII). Mucoid carcinoma was found as a patchy occurrence in some growths; the cells were scattered through areas of mucin which was supported in loose connective tissue. In one case the mucin development was such that the peritoneal metastases resembled psuedomyxoma peritonei.

FIG. XII. Photomicrograph of rat colon showing a carcinoma infiltrating the muscle wall and forming a subperitoneal nodule. Glandular and undifferentiated tissue are mixed together. (x 30)

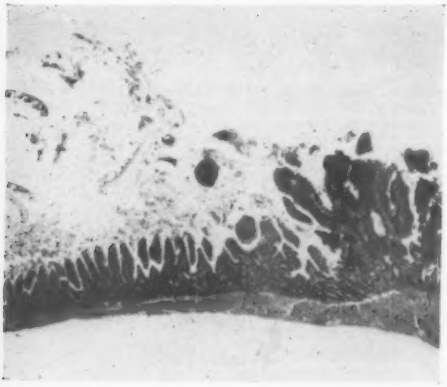


FIG. XIII. Photomicrograph showing hyperplasia of mucous membrane (at right). (x 9)

Numerous areas of macroscopically observed mucosal thickening (Figs. XIII and XIV), either localized or diffuse, showed various degress of enlargement and elongation of the glands. Maximal change often was found in the middle and basal layers of the mucosa though, in some, the superficial layers were also affected. Remarkable cellular proliferation occurred and, in most cases, numerous mitotic figures were demonstrable and the difficulty in distinguishing between precancerous and fully-developed neoplastic change was evident. In general, in these cases, the muscle layers were intact. That involvement of the muscularis mucosae indicated an early stage in the progress of the neoplastic process appeared to be demonstrated by the various stages observed in some of the examples showing multiple nodules.

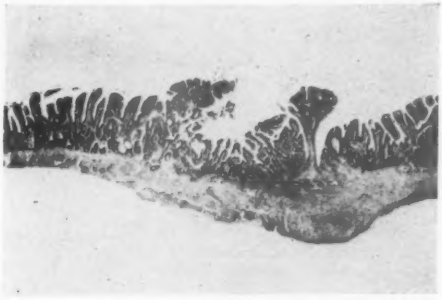


FIG. XIV. Photomicrograph showing glandular hyperplasia with beginning polyp formation. (x 8)

Fluorescein globulin staining

Frozen sections (of tissue snap frozen and cut in the deep freeze) were stained with fluorescein globulin complex. Normal tissues from various organs stained and fluoresced brightly in the usual manner.

All the tumour tissues failed to stain (in the way that has been observed with other animal and human tumours). In cases where tumour abutted on normal tissue, however, the clear demarcation usually seen was not always manifest. Much of the adjacent tissue fluoresced only slightly. At first it was thought that this might be due to some spoiling of the tissue by exposure to light during

preliminary photography of the specimen; such change has been observed to take place in the unfixed tissue on prolonged exposure. However, it was found that some areas did, in fact, stain.

Staining of portions of tissue showing the hyperplasia previously described, either localized or diffuse, demonstrated that some of these also stained poorly or irregularly. Examination of pieces of tissue from, as far as could be determined, unaffected areas showed that these stained and fluoresced as brightly as tissues from other organs. It was concluded, therefore, that the poor staining of the hyperplastic tissue was comparable with "loss" of staining observed in other experiments (Hughes, 1958) and due to its having undergone a "precancerous" change. This does not imply some vague predisposition but rather a definite cytological alteration in the direction of neoplasia. This conforms with the progressive nature of the condition as indicated by the predominance of indubitable tumours in the later stages of the experiment and the various stages found in examples of multiple involvement.

The comparison of the fluorescence picture with that of normal staining was obtained by fixing and staining a section with haematoxylin and eosin after it had been stained with fluorescein globulin and photographed with ultraviolet light (Figs. XV and XVI). Where complete absence of staining of a tissue corresponded with a typical histological appearance of neoplasia, the tumours (irrespective of spread) were regarded as true neoplasms.

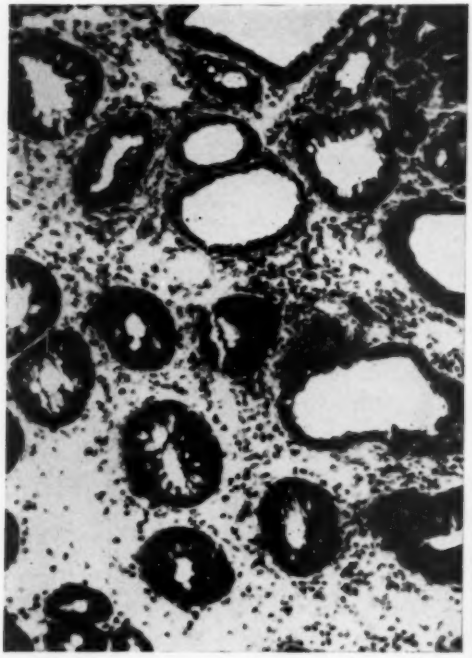


FIG. XV. Photomicrograph of the margin of a carcinoma of the colon; tumour alveoli above and to the right (Cf. Fig. XVI). (x 200)

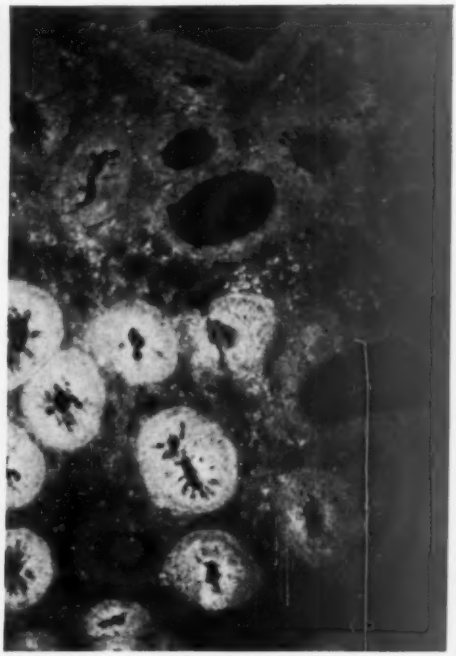


FIG. XVI. Fluorescence photomicrograph of the same section as Fig. XV; the tumour tissue does not stain and fluoresce but the normal glands do so. (x 200)

COMMENT

Apart from the general biological information gained, the study of experimentally in-

duced tumours is important, from our anthropocentric viewpoint, because of the information, direct and indirect, that it gives concerning the naturally occurring tumours of man. This investigation was part of a series covering a range of chemical carcinogens and a variety of tumours but it is considered separately because of the greater difficulty that has been experienced by almost all workers in producing bowel tumours.

The experimental method is of value for two reasons: first, once the technical details of the method and the appropriate materials are determined and obtained, it is possible to produce tumours at any time so that it is unnecessary to wait for examples to arise amongst numbers of other cases at autopsy or in the operating theatre. Of course it is not necessary, in this part of the world, to wait long for a naturally occuring carcinoma of the colon in man but, in general, only late stages of the condition are observed. Secondly, it is relatively easy to examine the tumours and their development at various stages of their formation since lesions may be observed or the animal sacrificed at any stage of the process. It has thus been possible to consider, in some greater detail than is usually practicable, many aspects of these tumours.

Carcinogens and their action

Of the many forms of chemical carcinogens, the types which have been shown, so far, to produce bowel tumours are some of the condensed polycyclic hydrocarbons and some of the aromatic amines. Relatively few tumours have been produced by use of the former, irrespective of the mode of administration and a series of experiments conducted personally has yielded negative results.

Current studies indicate that with many of the carcinogens it is not necessarily the material itself which produces the effect but rather some metabolite. These are definitely known in a few cases but in others the matter is still uncertain and is being investigated.

One feature, apparently common to most if not all the carcinogens, is the localization of the chemical material at the site where the effect is subsequently produced. In some cases the material is clearly incorporated in and apparently attached to some component of the significant cells; this has been shown particularly with members of the 4-Dimethylaminoazobenzene (butter yellow) group in the liver.

The significance of the various carcinogens is their probable relation to the development of "naturally occurring" tumours. This has been demonstrated in several types of carcinoma, especially some of those occurring in industry; indeed, it was the bladder tumours in workers with dyes that attracted attention first to the importance of the aromatic amines.

The specific action of carcinogens on one particular site in the body promises to provide an important clue to the nature of carcinogenesis. However, it has become apparent that, even when the particular material, whether it be the original substance or a metabolite, is demonstrated with certainty, other factors are important. Some of these which facilitate the action of the carcinogen are known as co-carcinogens and others which appear to inhibit their action are known as anti-carcinogens.

Although some carcinogens are remarkably specific in their action on certain cells or certain organs and even on those of a particular species, in some cases they act on more than one part of the body. This has been demonstrated in the present cases; not only is it clear that more precise knowledge of chemical characteristics, requirements and metabolism of individual cells is required but probably such localizations are influenced in some degree by the action of as yet unknown co-carcinogens.

The differences in the effects of various carcinogens in different species will be shown, in due course, to depend on the special chemical constitution of the cell in a particular animal as well as other factors which affect the general metabolism. The divergencies are shown by the occurrence, following administration of one of the Diphenyls, of bladder tumours in one strain of rat and intestinal growths in another. At present the differences are important since they emphasize that conclusions arrived at from the study of tumours in one animal must be applied to another with extreme caution. However if appropriate care is taken, information which may be valuable for the study of neoplasms in man may be obtained.

The special sex incidence which is quite remarkable in many cases is not shown to be significant in the present series of experiments; when present it emphasizes the influence of extraneous factors on the carcinogenic process. The presence of riboflavine in the diet inhibits the development of liver tumours with butter yellow feeding and this kind of effect may apply with other tumours; with the bowel growths, however, riboflavine appears to facilitate their formation (Morris, Wagner and Velat, 1955).

FIG. XVII. Photomicrograph of a transverse section of rat colon showing a tumour which has fungated into the lumen and also infiltrated the wall, forming a subperitoneal nodule. This caused intestinal obstruction. (x 15)

The long latent period, between the first application of the carcinogen and the appearance of the tumour, is common to all the well-known agents and when this period is considered in relation to the length of life of the animal it is found to correspond to the long period observed in man. This long latent period appears to be due to several factors. In the first place the processes taking place in the cell are complex and seem, in themselves, to require considerable time for the full development of the transformation. Secondly, the concentration of the material plays some part; but no matter how concentrated it may be the period cannot be cut down below a certain minimum, but with smaller or minute concentrations the latent period may be greatly increased. A further point is that ageing of tissue appears to play an, as yet not understood but, important part. This is clearly important in some circumstances where, on a group of experimental animals, some "naturally occurring" tumours

FIG. XVIII. Photomicrograph of a metastatic serosal nodule; the upper portion is mucoid in type. (x 30)

occur in the old controls. This of course means that significant influences which we do not yet comprehend are also active.

Characteristics of the tumours

The tumours corresponded very closely with those observed in man. The commonest growth was a fungating mass projecting into the lumen. In some cases it formed a definite polyp but in others it was sessile. Ulceration did not occur in a gross form but was present in small degree in many of the tumours. Predominance of ulceration with the formation of large craters, as often seen in man, was not observed. Local involvement of the surrounding tissues, in a concentric form,

sometimes gave a typical stricture; in other cases, infiltration along the wall gave the characteristic "garden hose" appearance. As with human tumours, the various types were sometimes combined (Fig. XVII). Spread occurred through the wall so that, in most cases, even the small tumours could be recognized from the exterior. Peritoneal spread resulted in distal serosal metastases (Fig. XVIII) and accumulation of mucus resembling pseudomyxoma peritonei was found in one case. More distant metastases have been described but were not observed in this series. Most of the features of the intestinal tumours of man were thus reproduced.

Microscopically, most of the tumours were of the typical adenocarcinomatous type but some of them were less differentiated and occurred as a relatively indifferent spheroidal cell type of carcinoma.

Most (four-fifths) of the tumours were found in the colon and, as far as specific sites were concerned, the majority was in either the rectum or the caecum with the former predominating. A few were present in the small intestine and one was found at the junction of the forestomach and the glandular stomach. Histologically the changes occurring in the mucosa were found, particularly in the early cases, in the mid and basal layers, some of the glands becoming dilated and others elongate and tortuous. In these there was a great increase in the number of cells, usually with an increase in the number of layers, so that the lumen of many of the growing buds was not apparent. There was the usual irregularity of cells and nuclei and mitotic figures were more numerous than is usually the case in human material. In cases where the whole thickness of the mucosa was affected, neighbouring areas showed the primary involvement of the deeper layers of the mucosa. The glands infiltrated the muscle layers and ultimately invaded the peritoneum.

This early involvement of the deep layers of the mucosa may be important from two points of view. In the first place it may be that, as in the skin, it is the growing cell which is affected by the carcinogen. Alternatively, it may emphasize that the carcinogen did not come to the area by way of the bowel lumen. Earlier, it was generally assumed that the localization of the carcinogenic activity was due to the site of application and, indeed, the paucity of result with administration of carcinogens by mouth was attributed to the inability of the materials to act on the mucosa because of mechanical interference by food and by their evanescence. However, recent information on the specificity of action of the carcinogens should throw doubts on this view and it is important that, even though local application (by feeding) of polycyclic hydrocarbons may produce occasional tumours (Rack, Kaufman and Simeone, 1955), the subcutaneous injection of these may also produce similar effects (Miller and Pybus, 1955). The action of the substances used in the present experiment (where all were administered subcutaneously) indicates that the effect is systemic rather than local, that is, the active principle came by the blood stream and was not applied locally. This conclusion is also evidenced by the occurrence of tumours in other regions including muscle.

A striking feature is the involvement of considerable areas of mucosa which, even if regarded as merely "precancerous," is of a kind patently related to undoubted neoplasia. In all cases there is considerable involvement of the mucosa in the neighbourhood of a growth and the multiple neoplasms also call attention to the general affection of the mucosa. This disposes of the question as to whether these tumours are of a unifocal or multifocal origin; all the evidence from these indicates that, within this one tissue, the carcinogen has a multifocal action. This corresponds well with the known characters of multiple polyposis and the associated carcinoma of the colon in man and goes a long way towards confirming some of the conclusions that have been drawn from observations on the naturally occurring disease.

Criteria for the diagnosis of malignancy different from those employed by the clinical pathologist have been used by workers on experimental carcinogenesis. It is understandable that in the experimental animal much more rigid criteria may be readily employed since, in human material, the patient is "given the benefit of the doubt" that there may be regarding the malignancy of any given lesion. The errors that have arisen in the past from the diagnosis of malignancy, when in fact it did not exist, have gradually been appreciated but, on the other hand, the

experimental worker has often gone to the other extreme. For example, it has been suggested that an epithelial growth should not be regarded as a carcinoma unless it has extended through all the layers of the intestinal wall and is apparent on the serosa (Stewart, 1953). One reason for the different point of view is the difference in size of animals. In the larger animals, including man, it is possible to recognize the characteristics of malignant invasion before all the layers of the gut wall have been invaded whereas, in the smaller animal, such as the rat, only a short distance separates the mucosa from the peritoneum and relatively gross invasion is likely to parallel cytological changes.

FIG. XIX. Photomicrograph of a frozen section stained with fluorescein globulin; tumour tissue (above) fails to stain and the normal glands (below) stain and fluoresce. (x 200)

At the same time the criteria of spread and invasion demanded by the experimental worker appear to be too rigid and, in any case, do not take into consideration that the development of malignancy involves essentially a change in character of the cell and that spread and metastasis are concomitants which, though almost invariable, are not necessarily an integral part of the process. Nevertheless, in the absence of clear indications of cellular differences at a cytological level, such criteria must still be important and, for the time being, it is desirable to accept the more rather than the less rigid criteria.

An important feature of the neoplastic change, just as it is following the application of carcinogenic hydrocarbons to the skin, is a modification of the muscle and connective tissue beneath the epithelium. In the early stages, the smooth muscle becomes thickened with cell proliferation and enlargement and these, with connective tissue cells, extend upwards towards the proliferating glands. To what extent this is a direct effect of the carcinogen or a development reciprocal with that of the epithelium is not apparent. At the same time it should be noted that neoplasms of smooth muscle type have not been found in these experiments. However, the contraction of tissue with an invagination of portion of the wall into the lumen, is an important factor in the formation of the polypoid processes.

As a result of these changes the epithelial glands may not reach the peritoneum until a late stage and some of these polypoid structures, which show all the general histological features of a carcinoma, may not necessarily be regarded as a malignant tumour by experimental workers. It is noteworthy that, in man, many metastatic intestinal growths are polypoid in form and, up to the time of death of the patient, are confined to the mucosal region. There seems to be little doubt that some of the experimental "polyps" are frank carcinomata but nevertheless the line of demarcation between the preneoplastic hyperplasia and indubitable carcinoma is difficult to make.

Fluorescein globulin stain

The necessity for some cytological means of diagnosing malignant tumours and possibly determining their early stages rather than depending on more gross histological changes is thus apparent. A method for distinguishing between normal and malignant cells had been developed and this was used in this experimental material. This method began as a presumably serological differentiation, a

γ-globulin being used as the specific agent. The serum globulin was obtained from animals (usually the rabbit) which had been injected with homogenates of normal tissue. This globulin was conjugated with a stain (the most useful being fluorescein) in order to illumine its site of action. Fluorescing dye was employed since this was the only type of stain which would give a sufficiently brilliant result to allow visualization of a minute amount of material. It was found that the globulin united with the protein of normal cells but not with that of malignant tumour cells.

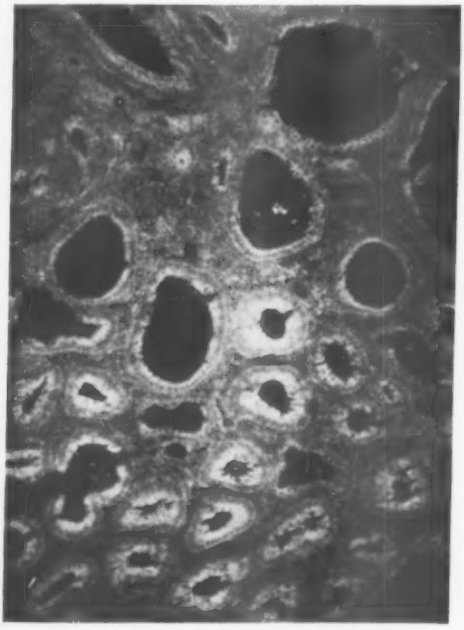


FIG. XX. Fluorescence photomicrograph of the junction of a tumour mass and histologically normal tissue. The differentiation of the two is poor due to poor staining of many of the glands (a few are fluorescing well). This is due to cytochemical changes (apparently of precancerous nature) not shown morphologically. (x 200)

It was then found that similar differential staining could be obtained not only by using globulin from rabbits which had not been injected previously with appropriate tissue (Hughes, 1957) but also from globulins of sera of other animals (King, Hughes and Louis, 1958). An important observation was that the same species, and even the same animal, could be used. Furthermore, not only γ-globulin but χ-and β-globulins and even albumins were equally effective (King, Hughes and Louis, 1959); this stresses further the non-serological character of the staining reaction.

These observations have been applied to a large series of tumours in animals (naturally occurring and experimentally produced) and also to tumours in man (Louis, 1957, 1958a, b). It was shown that innocent tumours stained in the same way as normal tissue, as did also most hyperplastic tissues.

FIG. XXI. Photograph of portions of colon removed at operation, four separate and apparently independent carcinomata (associated with multiple polyposis being present).

At the same time, it was found that, in experimental animals in which hepatomata had been produced, groups of cells showed a loss of staining prior to the development of frank neoplasms (Hughes, 1957). These "islands of loss" of staining were interpreted as being a "precancerous" change; a similar loss of staining was observed in polyps of the colon; they were found in multiple polyps but not in the single polyp (Louis, 1957). Similar changes were found in mammary ducts in cases of carcinoma (Louis, 1958b).

In the present cases tumours all failed to stain and the normal tissues did so normally. In some cases there was the sharp demarcation

of the two as observed with other kinds of neoplasm (Fig. XIX) but in others the tissues surrounding the growths did not demonstrate the characteristic difference (Fig. XX). In many areas this failure to stain (or diminution in staining capacity) was present away from recognizable tumours.

This means that, though the well-developed growths are clearly differentiable from normal tissue, areas of non-neoplastic tissue not so clearly separated from the tumours are present; probably these represent "precancerous" tissue. Thus this staining method is not applicable, in its present form, as a means of determining precise cytological characteristics of the early stages of the tumour growth, almost certainly because these are at the biochemical and submicroscopic level; it does appear to delineate, however, the area of tissue which has been affected by the carcinogen and the presumable (and, in some cases, the observed) site of subsequent tumour formation. It demonstrates that these growths and their immediately related tissues correspond with the naturally occurring and preneoplastic hyperplasias of man.

This emphasizes once more the wide zone of action of the carcinogen and suggests that the condition of multiple polyposis in man may be due to some such chemical agent; moreover, the multiple growths are similar to and probably comparable with the naturally occurring occasional multiple tumours of the human colon (Fig. XXI). Furthermore, the observations of the kind presented here also indicate that we do not necessarily have to look for a dietary carcinogen as the cause of cancer of the colon. Irrespective of what conclusions may justifiably be drawn from these few observations, it is apparent that we have here a valuable means of studying an experimentally produced tumour which closely resembles in many ways the carcinoma of man.

Surgery in this country owes a great deal to Sir Hugh Devine and his contemporaries. The transformation, in the last half-century, of the lot of sufferers from conditions which are now amenable, in some measure, to surgical treatment is due, in no small degree, to their example and precept in the application of basic principles to everyday therapy. It is in salutation to him that this tribute is presented.

SUMMARY

Carcinoma of the bowel can be produced readily in rats using an aromatic amine. A study was made of a group of examples which developed following subcutaneous administration of 3:2′-Dimethyl-4-aminodiphenyl and 3:3′-Dimethyl-4-aminodiphenyl. The tumours occurred particularly in the colon though some arose in the small intestine and one in the stomach. Diffuse hyperplasia of the mucosa occurred commonly and multiple growths were found on several occasions. Complications such as obstruction, early ulceration (with haemorrhage) and intussusception occurred. The staining characteristics of the tumours with fluorescein globulin corresponded with those of other malignant neoplasms. Areas of "loss of staining," indicating a "precancerous" development, were common.

ACKNOWLEDGEMENTS

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THE BEST TECHNIQUE IN GASTRECTOMY FOR ULCER

THE SEARCH FOR AN IDEAL

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Sydney

THIS search by many seekers began eighty years ago, when a young surgeon sat helplessly by the bedside of a man dying from pyloric obstruction. He asked himself: "Need this happen? Cannot something be done about it? After all, my master has resected a carcinoma of the oesophagus. Why not also of the pylorus?" In medicine, pity does not rest content with its own tears. A profound emotional disturbance moves the will to action, and sometimes the dynamism goes on beyond immediate succour, even to new discoveries; and, thus, new methods are born.

FIG. I. Carl Gussenbauer (1842-1903).

The young man was Carl Gussenbauer (Fig. 1) and his master was Theodor Billroth, working in the great *Allgemeines Krankenhaus*, once the *Grosses Armenhaus* in Vienna, where for centuries the sisters had nursed the indigent and sick of Central Europe and of the Balkans (Fig. II). Here, Rokitansky, following the example of Morgagni, had carefully studied and recorded autopsy findings on a colossal scale, teaching that medicine could be soundly based only upon a thorough knowledge of pathological anatomy. He did not lack material, for the Emperor Joseph II had made autopsy permission a condition of entry to the enormous hospital. And so, the young man, with the help of his friend, Von Winiwarter, was able to study instructive autopsy reports, back to Napoleonic times.

FIG. II. The Alserstrasse, Vienna, showing the Allgemeines Krankenhaus on the left.

And in these he found further encouragement, for many patients had died without metastases, and with primary growth still free. But surgeons feared for the healing power of the stomach. Could it withstand the corrosive and digestive action of the gastric juices? Would not disaster follow from gangrene, perforation and peritonitis? And, later, from ulceration and stricture? And so we find the curious paradox that the way was paved for the simpler operation of gastrectomy by the more difficult and hazardous one of oesophagectomy.

In February, 1874, Gussenbauer and Von Winiwarter performed the first partial gastrectomy upon the first of a series of dogs, removing the antrum and pylorus, and suturing stomach and duodenum end-to-end. The dog died of peritonitis, but survived long enough to show that the inverted serosal surfaces at the suture line adhered firmly, and the mucosal edges remained healthy. Gussenbauer attributed the peritonitis to the septic environment and instruments, for he had operated in the autopsy room. Vienna had at last learnt to appreciate the gospel which Semmelweiss had preached there in vain during his lifetime.

Seven years later, in this same room, Billroth witnessed the autopsy upon a woman upon whom he had done his first partial gastrectomy, three and a half months previously, and who was now dead, with hepatic metastases. The liver is still preserved. And here, in this huge, dark and sombre old room, with many autopsy tables, I did my own first gastrectomy, assisted and tutored by Leopold Schönbauer, who later, in his turn, became Professor of Surgery in this old University.

Gussenbauer and von Winiwarter found that the feared complications did not beset the anastomoses in their dogs, and they argued, from Gunther's successes in gastrotomy for foreign bodies, and from the results of accidental wounds, that wounds in the human stomach would also heal. Their paper (1876) attracted the immediate interest of Continental surgeons, and each school hoped for the honour of salvaging the first patient. Péan's patient in 1879, and Rydygier's in 1880, failed to survive the operation. Billroth's first operation was done on 29 January, 1881. When it was seen that the patient was surviving, Billroth (at the invitation of Dr. L. Wittelshöfer, Editor of the *Wiener medizinische Wochenschrift*), wrote on the 4 February a letter describing the operation, and answering critics who had described the attempt as foolhardy. This letter was published in the number of the journal which appeared on the following day.

In France, the operation was called after Péan, but in most countries it is called after Billroth. Perhaps Gussenbauer-Billroth would be more just. Even von Eiselsberg, loyally defending his own beloved master, does scant justice to Gussenbauer (1929). Billroth was not only a great surgeon, but he had a charming and magnetic personality, which endeared him to his associates. Even today in Vienna a street commemorates him, and, in 1929, the centenary of his birth was celebrated by a great feast and ball, given by the Burgermeister in the magnificent Gothic town-hall, and attended by surgeons from all over Europe. In addition, a two-shilling piece was struck in his honour (Fig. III). Nevertheless, Gussenbauer has earned our remembrance.

Fig. III. The Austrian two-shilling piece struck in honour of Christian Albert Theodor Billroth (1829-1894).

For simple ulcer, which concerns us most in this communication, the first gastrectomy was done by Rydygier (Fig. IV), in a small private hospital in Kulm (Chelmo), Poland, also in 1881. The patient was shown, in good health twenty-five years later, at the German Surgical Congress in 1906.

During the next thirty years, the German schools were very interested in gastrectomy and busied themselves testing the many possible modifications of Billroth's two basic methods. This was natural, because so many chairs of surgery were filled by Billroth's assistants of the first and second generations, and not only in German-speaking lands. As Woodward (1958) has recently reminded us, von Eiselsberg, a Catholic, overcame the barriers of religion and language, to take the Chair in Utrecht. Perhaps the Dutch had learnt their lesson, after losing de Graaf and

Van Swieten. Billroth, himself a Prussian and the son of a poor Protestant clergyman, from the Baltic Island of Rügen, had been received with open arms by Catholic Vienna. And, as if to show the transcendence of medicine over national and religious barriers, there was never a more fruitful co-operation in surgery than that between the master, and the band of brilliant young men, who were soon to dominate the surgery of a continent. Billroth was ever keen to help the careers of his assistants, and it is of interest to read

FIG. IV. Ludwig Rydygier (1850-1920).

in his letters, not only his fears about von Eiselsberg's appointment to Utrecht, but also his satisfaction that the Catholic, von Mikuliczs, at least, will not have similar difficulties in Poland. But it is my present task to pursue not the history of a school, but the story of a technique.

The technical problems are those of selection. Each step in the operation has been done in so many ways that the various possible combinations amount to hundreds. But now, after eighty years, we should have sufficient facts and knowledge of principles, to ensure selection of the best technique. For each step, there is only one best way. Sometimes the next best is not far behind, but sometimes it is so defective as to jeopardize the health and life of the patient.

At the beginning of our search, we should remember that operative technique is only one of the essentials. The others are the proper selection of patients, a proper physiological and psychological preparation and after-care, and the proper selection of operation for the patient.

SOME PRINCIPLES OF TECHNIQUE

Even when the best of technique is chosen, failure may still follow, for a good technique can be badly applied. In gastric surgery, precision cannot be over-emphasized—for even a small haematoma, a slight misplacement of the jejunal loop, the insertion of a suture a few millimetres to the right or left, a little bruising and impairment of capillary circulation, may bring failure.

Care and gentleness, the minimum of tissue trauma, patience and persistence, perfection in haemostasis, precision in reconstruction and suturing—these are the watchwords. They are not new, but can ever bear repetition, especially when illustrated in the work of the great masters. And I can do no better than begin with Hugh Devine. For we can truly say of him what Fulton said of Cushing—"In all he did he was a perfectionist." In watching many surgeons in many lands, I found that the method of open anastomosis and detailed haemostasis used by Devine was unsurpassed, and this I have tried to imitate. And now Hollenbach (1950), reporting his magnificent series, one of the best from the German schools, describes and stresses the method of haemostasis and suturing, and it is essentially the method proposed and practised by the Australian master so many years previously. The cut edges to be co-opted are the critical tissues of the operation. The life of the patient in the immediate post-operative period, and, later, his freedom from stomal ulceration, depend upon their anatomical and biological integrity. The life-line upon which they depend are the capillaries and delicate vessels passing to and from them in the visceral walls. Devine also reminds us that the mucosa glides freely on the subjacent muscularis, and a destruction of this mechanism will make the tissue more vulnerable to the

traumata of daily work. The sub-mucosal areolar tissue may be destroyed by abscess, or by low-grade injection, collections of blood or serum and by rough handling or crude through-and-through suturing, all of which tend to excessive inflammatory reaction and swelling in the immediate post-operative phase, and to excessive fibrosis in the later stages, with poor thin epithelium, covering a fixed scarred stomal ring. The so-called "non-crushing" clamps are abhorrent to Devine, and his followers. Devine (1940) pointed out that "there is only one sure way of attaining exact haemostasis, and that is to avoid the use of bowel clamps." Hollenbach described and illustrates from work done in the experimental animal the destruction wrought by these clamps—the crushing of the mucosa, the rupture of vessels and haematomata in the other layers, thus ensuring some obstruction to the arterial supply and to the lymphatic and venous drainage, the very life-lines of the healing edges. When the clamps have been applied, at their best the linear mark which they make almost disappears on the seromuscular surface, but the crushing of the mucosa remains visible, and the microscope shows the damage caused to the muscularis. If such clamps are left on during the suturing, inspection, just before the abdomen is closed, will show that the tissue between the clamp line and the stoma is more swollen and succulent and of a deeper colour than the tissue on the other side of the clamp line. I have little doubt that this reaction goes on, and becomes intensified after the operation, sometimes causing gastric retention. When the open method of operating is used, the field should be carefully packed off, and I point this out because I have been horrified to watch surgeons abandon this precaution, and leave the open stomach and subphrenic spaces exposed in the same field, so that gastric contents and blood trickled from one to the other, as though subphrenic infection were deliberately sought after. It is not sufficient to rely on the suction nozzle for the removal of this fluid, because it reaches many tiny nooks and crevices among the viscera and folds of omentum, which are missed by the suction nozzle.

Although the method of open operation and painstaking haemostasis is time-consuming, Devine was by no means a slow operator. But he preferred perfection to speed, and he was at one with the surgical revolution led by Kocher and Halsted, who had broken with the more "bloody, bold and resolute" traditions from pre-anaesthetic days. When the young Cushing went from Halsted to Kocher he wrote "curious how much Kocher is like 'the Professor' (Halsted), careful, painstaking work, elaborate technique, and all the rest which we have in Baltimore." And again, "But the operating! the J.H.H. (Johns Hopkins Hospital) outdone. It's easily seen why 'the Professor' thought so highly of their work. Detailed technique, tedious operating, absolute haemostasis" (Fulton 1946). In their turn, Cushing's long operations, lasting hour after hour, displaced the much more rapid methods of Sargent and others.

FIG. V. Anton von Eiselsberg (1860-1939).

Leriche (1956) became a disciple and wrote just before he died, "personally, since I saw Halsted and Cushing, I no longer went for speed. Their technique was so very superior in its results to what I had hitherto seen! It achieved such safety that it imposed itself on every unprejudiced onlooker. In fact, the essential thing is to be a safe operator. I have aimed at this by precision in act, gentleness of hand, care in haemostasis, and by a minuteness of reconstruction. Is it not quite absurd to let the patient bleed and then

to transfuse under the guise of 'replacement'? It is so much more in accord with the science of the living, to avoid blood loss! Gentleness and haemostasis alone permit the achievement of all surgery at all levels."

In surgery of the breast, Haagensen and Stout (1942) found that the radical operation taking four hours had twice as many survivors, and half as many recurrences at the end of five years, as compared with the operations lasting but one hour.

FIG. VI. Hans Finsterer (1877-1955).

In gastric surgery, Finsterer also was painstaking and slow, taking four hours for a simple gastrectomy, up to six, if complicated, for example by gastro-colic fistula. His careful technique enabled him and his friend, von Eiselsberg (Figs. V and VI) to achieve a mortality of 2½-3 per cent. in gastrectomy, including penetrating ulcers, while surgeons in other lands admitted to a mortality of 19 per cent., even in non-penetrating ulcers. Later, at an Austro-Swiss Medical Congress, Finsterer (1950) reported a 2.9 per cent. mortality in 2,472 patients, including those operated on before the days of sulphonamides and antibiotics and easy transfusions, and those suffering from the huge ulcers with malnutrition in the famine days of two world wars.

I am aware that there are surgeons with rare gifts of dexterity who use clamps and continuous haemostatic stitches, and still achieve good results. But it is clear that their results could be still further improved by a more refined technique whereby tissue trauma is lessened, and haemostasis made more secure. I am afraid that some of our young surgeons, who bring back from overseas the old clamp and haemostatic suture prefer speed and brilliance to the perfection of technique in gastro-intestinal surgery which Hugh Devine had introduced in their own country. I am at times moved to recall the words of Moynihan (1920) in the first J. B. Murphy oration — "There are still among us brilliant operators from whom I pray to be spared when my hour has come. For them it is the mere quality of effort that counts. Their idea of operative surgery is something swift and infinitely dexterous, something to dazzle the beholder and excite his wonder that such things can be done by human hands. The body of a man is the plastic material in which an artist works, and no art is worthy of such a medium unless it has in it something of a sacrament. Surgery of the 'brilliant' kind is a desecration. Such art finds its proper scope in tricks with cards, in juggling with billiard balls, and nimble encounters with bowls of vanishing goldfish. But Murphy was of the true faith. He believed in safe and thorough work rather than in specious and hazardous brilliance. He was infinitely careful in preparation, and, compared with many, was inclined to be slow, but every step in every operation which I ever saw him do was completed deliberately, accurately, once for all. . . . And so, when the end came, a review of the operation showed no false move, no part left incomplete, no chance of disaster; all was honest, safe, simple; it was modest rather than brilliant."

SOME DETAILS OF TECHNIQUE

The incision should be median, preferably stopping above the umbilicus to preserve the strong musculo-aponeurotic sling which crosses the abdomen just above the umbilicus, and which is marked externally by a sulcus, easily seen upon the male abdomen (Fig. VII). Transverse or paramedian incisions do

not utilize the subcostal angle to best advantage for upward access. The paramedian, moreover, requires the suturing of the posterior layer of rectus sheath, the fibres of which allow the sutures to cut-out readily. The cutting-out may not be noted, because of relaxants. It awaits the first cough after the skin is sutured. The linea alba, on the other hand, does not readily allow cutting-out, and, moreover, it presents but one layer for suture.

If upward access is difficult, the parietal incision should be further extended rather than risk imperfections in visceral suturing. To this end, the incision is extended over the left costal margin, the rectus muscle is divided on the chest wall and turned down, and costal cartilages and xiphisternum are removed as in the old methods of Marwedel and Pauchet. I have also mobilized the costal margin by dividing the cartilages in small separate wounds. This is far better than adding the hazards of thoracotomy to those of laparotomy.

The full thickness of the wound edges are then wrapped in heavy gauze towels, held in place by the Devine self-retaining retractor. The towels protect the wound from the skin surface which is a main source of staphylococcal infection and from infected material from within the abdomen. They also prevent the tiny trickle of blood which sometimes continues unnoticed, from the wound edges into the complex recesses of the peritoneal cavity, there to remain, adding to the immediate post-operative peritoneal reaction and increasing the risk of gastric retention, paralytic ileus, sub-phrenic abscess and, later, adhesions. The steadiness of this retraction is preferable to the repeated tugging and releasing of manual retraction, for, to minimize shock, afferent stimuli must be minimized. Adaptation takes place to the steady stimulus. Impulses are set up at the "make and break", as the student knows from his muscle-nerve preparations, and as the anaesthetist knows from his observations on airways and intratracheal tubes. The surgeon who opens the abdomen under local anaesthesia knows that even when the anaesthesia has partially disappeared the steady self-retaining retractor is well-tolerated, but the attempt to remove it would cause a painful straining and sweating, if a re-injection were not given. And the nursing sister soon learns to expect shock in the patients of the terrier-type surgeon, who vigorously tugs the tissues and organs hither and thither.

The freeing of the greater curvature should be done by dividing the small vessels between the gastro-epiploic trunks and the stomach, sparing the main vascular arch, which provides the blood supply of the omentum. The omentum cannot well exercise its police or defensive functions, if devitalized. Its devascularization may turn it into an

FIG. VII. The abdominal girdle.

oedematous mass, later to become a fibrosed adherent ball. (The curvatures must not be freed to the pylorus, if Finsterer's gastrectomy with exclusion is to be done.)

The duodenum is then divided, and its distal end closed. This is best done by placing a Payr clamp across the antrum as a holder, and making a circular incision around the duodenum, dividing only the sero-muscular layers which are pushed back like a cuff, and the mucosa is divided more distally. The sero-muscular cuff, unbruised and well vascularized, is then closed over the unsutured mucosa by a running stitch of fine catgut. This must be done so perfectly that no other suture is necessary. This first suture line is

then inverted by fine silk Lemberts. The reinforcement by tags of omentum is quite useless to prevent a leak, if the mucosa is not buried. This, the best of the many methods of duodenal closure, I learnt from Devine.

The gastro-jejunal anastomosis should be ante-colic. This method is easier than the retro-colic, eliminates the obstructive hazard of the mesocolon, and allows easier re-operation if this should be needed. The jejunal loop should not be so short as to obstruct the colon, nor so long as to allow another loop to pass behind it. There is no room for inexactness here. As the first loop is brought forward the colon slips backwards to occupy the space vacated, and post-operative skiagrams, antero-posterior and lateral, show that there is no looping of the afferent limb, as it passes upwards in a straight line in front of the transverse colon to the upper end of the stoma. A second reason for avoiding a long afferent loop is that the incidence of stomal ulcer increases directly with the distance of the stoma from the duodeno-jejunal flexure. The stress in the recent literature upon the obstructions due to the long ante-colic loop is merely another illustration of the principle that the bad results of gastrectomy result from bad gastrectomies.

The afferent jejunal loop should pass to the lesser curvature of the stomach, because gastric systole, aided by gravity, will then empty the food into the efferent loop, and not in a retrograde direction, towards the duodenum. The gastric and jejunal streams are thus made to converge at an acute angle. Confluence rather than turbulence is secured. Turbulence has an adverse effect upon the containing walls of any stream. In addition, it delays emptying and prolongs the digestive phase in the region of the stoma, and tends to mucosal inflammation.

So the emptying should be stream-lined. The founders of gastric surgery recognized this requirement. If the afferent loop is placed to the greater curvature, the *anastomose inversée* of French authors, turbulence and delayed emptying result; "dumping" symptoms and stomach ulcers become more common.

The line of incision in the jejunum should lie precisely on the anti-mesenteric border. If it is oblique, torsion and obstruction and "dumping" symptoms result. The line of incision in the gastric wall should be slightly oblique, passing downwards and to the left, so that the jejunum passes in a straight line from the lesser curvature, downwards and to the left, into the efferent limb, avoiding the "question-mark deformity" of Visick.

The method of suturing has been partly described. For the outer sero-muscular layer, interrupted Lembert sutures of fine black silk are used, and tied to produce apposition, but not tearing nor blanching. Interrupted sutures provide a more precise apposition of rolled surfaces than do running sutures. Those first placed act as holders, and, if inversion is difficult, they are all passed before they are tied, and then held up together, to distribute the strain, so that an inversion can be more safely accomplished. The sero-muscular coats of stomach and jejunum are then incised down to the sub-mucosa and the vessels in the gastric sub-mucosa are under-run with fine catgut sutures and ligated, and those in the jejunal sub-mucosa are divided between fine forceps and tied. An inner suture of fine catgut then unites the cut edges of the sero-muscular coats. The mucosa is left unsutured. Suturing traumatizes it, and does not produce an edge-to-edge apposition, nor a leak-proof suture line. It adds nothing to the security of the anastomosis. The mucosal edges lie more neatly if allowed to rest as flaps, lying on the muscular coat. Collections of serum, blood or pus can then erupt more safely towards the lumen.

Recently, the Hungarian surgeons, Török and Karlinger, have performed gastrectomy on dogs, studying the healing of the anastomosis, endoscopically and histologically. They found that the inclusion of the mucosa in the suturing was followed by a severe and prolonged thickening and inflammation in the neighbourhood of the stoma, with second-intention healing, and later, a coarse, hard and rigid scar. When the mucosa was left unsutured, to lie as a free flap, the "anastomositis" (as they call it) was much less severe and prolonged, and the resulting scar was "much finer, nowise hard, but plastic". They thus confirm the earlier findings of Hollenbach. They explain that suturing the mucosa violates the principle of providing free drainage in the repair of contaminated wounds.

The operation thus far accomplished, with ante-colic jejunal loop, afferent limb to lesser curvature, and full-width stoma, was first used

on 24 Nov. 1885, by Krönlein of Zürich (Fig. VIII), and is properly called the Krönlein modification of the Billroth II. It was the second operation done by the Billroth II method. Ten months previously, Billroth had anastomosed the stomach and jejunum side-to-side. The third operation by the Billroth II method was done on 2 April 1888 in the Billroth Clinic by von Eiselsberg, who acted on the suggestion of von Hacker (pronounced "Harker"), and closed the upper half of the stomach opening, using the lower half for end-to-end anastomosis with the jejunum.

FIG. VIII. Rudolf Ulrich Krönlein (1847-1910).

At the lesser curvature end of the stoma, the apex of the jejunal loop forms rather an acute angle, which tends to a minor degree of obstruction. This is prevented by the stitches first described by Kapeller (1898) which suspend the afferent limb to the stomach above the stoma, so that the antimesentric border of the jejunum is lifted away from the mesenteric border, thus widening the lumen at this angle. These stitches also add to the security of closure at this, "the danger point". These stitches must be so passed that the anterior and posterior surfaces of the jejunum are fixed to the corresponding surfaces of the stomach. The temptation must be avoided to fix the apex of the jejunal angle to the anterior surface of the stomach, for this would produce an obstructive torsion. A minor degree of obstruction at this point, due to an omission of these sutures, or to their improper placement, or to some other fault, is a common cause of "dumping" symptoms and bile-vomiting.

The next step to minimize obstructions is the placement of the efferent loop so that it passes downwards and to the left, away from the greater curvature without kinking, and the omentum is then so placed that it intervenes between afferent and efferent loops, and also between the jejunum and the parietal incision. The abdominal wall is then closed in accordance with the expectation that the patient will cough vigorously in the post-operative period. Post-operative wound disruptions and herniae are not excusable on the grounds that the patient coughed. After gastrectomy, coughing is to be expected.

After operation, as soon as the patient can swallow, drinks are given, 1 oz., hourly, water, tea with lemon, fruit drinks, strained broths, or Vegemite (yeast-extract) drinks, but no milk. These are sipped slowly. If well tolerated, they are increased to 2 oz. at the end of six hours and to 3 oz. at the end of twelve hours. Meantime the stomach is aspirated four-hourly through the indwelling plastic tube, made from the tubing from blood-transfusion sets, and swallowed before operation. When the stomach is clearly passing on its fluid contents, usually after twenty-four or forty-eight hours, the tube is removed. Intravenous fluids and blood transfusions are rarely needed. The patient gets out of bed on the second or third day, and leaves hospital on the twelfth. It is explained to him that during the stage of healing he must give reasonable consideration to the stomach, and "take the strain off it". This means small frequent meals of soft foods, eaten slowly, thoroughly masticated, and taken dry, with fluids between meals, and lying down after each meal. These restrictions are gradually relaxed, except the slow eating and thorough mastication.

THE HIGH GASTRIC ULCER AND THE "BEUTEL-MAGEN"

For a high gastric ulcer, the Hacker-Eiselsberg method is used, slightly modified in that the upper part of the gastric incision does not continue in line transversely across the stomach to the lesser curvature, but curves

upwards towards the right border of the oesophagus, thus removing the lesser curvature. This part of the incision is closed, thus forming a tube of the greater curvature, which is anastomosed with duodenum or jejunum. This modification was first described by Schoemaker of the Hague in 1911, and also by Pauchet and Kirschner. It is called the tubed (*Schlauch*), or step-shaped (*treppen-förmig*) resection.

When a lesser curvature ulcer, irrespective of its distance from the pylorus, fore-shortens the lesser curvature, the stomach ultimately comes to resemble a tobacco-pouch (*beutelmagen*). In such cases, even though of only moderate degree, the Hacker-Eiselsberg or Schoemaker method should be used, because the full-width stoma in these cases tends to minor obstruction at the upper point of the jejunal loop. Another indication for tubed-gastrectomy is the short jejunal mesentery.

In 100 consecutive gastrectomies for gastric ulcer I have used this method 44 times.

THE FIXED PENETRATING DUODENAL ULCER

Recently accounts have appeared in the surgical literature of injury to the bile and pancreatic ducts through injudicious efforts to excise duodenal ulcers penetrating the pancreas, thus affording further illustration of the thesis that the bad results of gastrectomy are the results of bad gastrectomies. Such ulcers should not be excised, but should be dealt with by one of two methods, the choice of which depends chiefly upon the condition of the anterior wall of the duodenum, partly upon the build of the patient.

If the anterior wall of the duodenum is mobile, and freely accessible, the division is made through the proximal edge of the ulcer, and closure is made by rolling the anterior duodenal wall into the ulcer, *en escargot*, using the methods described by Bsteh, Nissen and others. This operation is called gastrectomy "with duodenal exclusion", or "with deep exclusion".

If the anterior wall of the duodenum is so diseased that it will not readily roll in and perhaps inaccessible through the sthenic habitus of an obese patient, Finsterer's gastrectomy with exclusion is done. The right gastric and gastro-epiploic arteries must be left undisturbed on the pyloric antrum. The stomach, held by a Payr clamp, is divided distal to the clamp down to the sub-mucosa, two or three fingers breadth from the pylorus, and the antral mucosa is removed. As the sero-muscular cuff is turned back the vessels passing from it to the mucosa are clamped in fine forceps before division, and tied. The mucosal core is divided at the pylorus and the opening closed by a continuous catgut suture. The sero-muscular cuff is then closed by a continuous suture of fine gut inside the cuff, uniting its anterior and posterior walls. The edges of the cuff are then united by interrupted fine silk sutures. Inversion of the cuff should be avoided, because the cuff is broadest at its cut edge, and inversion tends to capillary obliteration and necrosis. Quist (1958) has recently excised wedges from the anterior and posterior walls, fashioned a cone from the remaining curvatures, and inverted the cone. Finsterer advised drainage to the excluded pylorus — I do not do this.

In my last 100 consecutive gastrectomies for duodenal ulcer, I have used the classical closure of the duodenum 50 times; deep-exclusion 40 times; and pyloric-exclusion 10 times. As experience increases, deep exclusions tend to replace pyloric-exclusions. Results have been good by all three methods. The only early complication was a minor fistula following a pyloric-exclusion, which closed spontaneously.

FINSTERER OR BANCROFT?

I have recently pointed out that "gastrectomy with (pyloric) exclusion" was introduced by Finsterer in 1918, and that to call the operation after Bancroft is unjustifiable. Since then, Quist (1958) has re-opened the question of priority and insists on calling the operation after Bancroft. The following review of the facts should finalize the matter.

In 1895, von Eiselsberg described a method of dealing with irremoveable pyloro-duodenal masses, most of them carcinoma, by dividing the stomach proximal to the mass, and doing a gastro-enterostomy in the proximal segment. The principle of the operation is mechanical, diverting the food stream through a non-ulcerated, non-obstructed channel. Devine described a similar method. Early results were good, and the operation was later done for known duodenal ulcers. It was soon discovered that results in duodenal ulcer were very bad, because of the later development of stomal ulcer. Failure to deal with the acid

factor was the reason for this, and it was recognized by Finsterer, who, therefore, made the operation a gastrectomy.

In 1918, Finsterer wrote: "The fact that peptic ulcer can arise at the stoma, not only after simple gastro-enterostomy for duodenal ulcer, but also after the unilateral exclusion of von Eiselsberg, led me to excise half to two-thirds of the stomach in all cases, not only when the ulcer is resected, but also when it is excluded, in order that the hyperacidity should be considerably reduced, by reduction of the area of secreting mucosa. . . . The small stomach stump is closed blindly after excising as much as possible of the mucosa . . . The scientific explanation of the disappearance of the hyperacidity is provided by the experimental researches of Edkins." Edkins had proposed the gastrin theory in 1906 and 1908.

From 1918 onwards, Finsterer published many further reports of the results of his operation, in the German, French and English literature. So also did many other surgeons.

In 1932, Bancroft first came on the scene, describing "a modification of the Devine operation of pyloric exclusion for duodenal ulcer". He proposed a coning out of the antral mucosa "as a means of correcting the aforementioned disadvantages of the Devine operation," namely: "(1) there is left a gastric mucous membrane in the distal portion of the stomach which may continue to bathe the ulcer with its secretion, and thereby delay healing, (2) it is generally considered an inadvisable surgical procedure to leave a blind pouch anywhere in the gastro-intestinal course." Bancroft mentions neither stomal ulcer, nor acid. His proposed reasons (given above) may perhaps be questioned. The external secretion of the antral mucosa is bland, alkaline and mucoid, and should do more good than harm, if it "bathes the ulcer". And, as for the blind end, if he is speaking anatomically, coning out the mucosa does not remove the blind pouch, but merely moves its closed end a little distally. If he is speaking physiologically, there is no blind antral pouch because the stream is moving, not towards the closed end, but away from it, as he says, bathing the duodenal ulcer with its secretion.

In 1951, Bancroft wrote a misleading article, quite at variance with that published in 1932. Now, for the first time, he mentions acid and hormone. The operation which he modified in 1931, and which he then called "the Devine operation of pyloric exclusion", he now calls "the Finsterer operation of pyloric exclusion". Bancroft then writes: "Since the original description in my paper (of 1932), I have modified my procedure in that I resect the middle third of the stomach". In other words, Bancroft now, in 1951, adds to "the Finsterer operation of pyloric exclusion", the gastrectomy which Finsterer had added in 1918! And in reporting the work of twenty years with "his procedure" he mentions neither the number of his patients nor their mortality.

In 1958, Quist wrote as follows:—"In 1932 Bancroft described exclusion gastrectomy with 'coning out' of the antral mucosa as a deliberate and important step in technique, and established the operation on a sound physiological basis."

The fact is that the operation described by him in 1932 was not a gastrectomy and the reasons, or physiological basis proposed by him, are seriously open to question.

To displace Finsterer's name from the operation of gastrectomy with exclusion, and to substitute Bancroft's, involves a travesty of truth and a denial of justice.

EARLY RESULTS

I have now done 369 gastrectomies for simple ulcer, including gastritis. In 335 interval cases, including 13 who had had previous gastro-enterostomy or inadequate gastrectomy, there have been four deaths, described in a previous communication (1956), a mortality of 1.2 per cent. None of the deaths was due to a failure of the technique described. In the last 260 consecutive cases there have been no deaths. Among 33 patients operated on during acute bleeding there have been four deaths. One patient, operated on for acute perforation, survived.

The immediate morbidity arising in the operative site has been minor and transitory. Since my previous communication, one patient has had a post-operative haemorrhage, which stopped spontaneously after a transfusion of 1 litre of blood. Another, mentioned above, had a minor fistula from an excluded pylorus which closed spontaneously.

The technique chosen *à priori* has justified itself in practice and can be recommended with confidence.

STOMAL ULCER

Since my publication in 1956, which reported 6 stomal ulcers, I have seen one more, Mr. J. D., whose symptoms reappeared ten years after his original operation. He is my first patient to acquire a stomal ulcer after an interval primary gastrectomy according to the technique described above. His primary operation was a gastrectomy with exclusion. The antral mucosa was removed, but the gastric resection was insufficient. The patient is now symptom free, following further gastrectomy with resection of the jejunal loop.

In stomal ulcer, surgeons have rightly directed their attention to the acid peptic juice. But this point of view should be supplemented, as the following case shows:

Mrs. J. McA., aged 64, had digestive symptoms with a filling defect at the pylorus due to antrum gastritis. She was averse to operation, and, with some anxiety, we kept her under observation from. 1951 onwards. In 1956 she developed severe pains, and a large niche typical of simple ulcer, high on the lesser curvature, just where I would have divided the stomach if she had come to operation.

In duodenal ulcer, also, as the years go on and acidity lessens, there is a tendency for the ulcer to heal, and for a new one to appear on the lesser curvature of the stomach. Tanner (1954) has stressed this.

It is, therefore, possible that some "stomal" ulcers are really gastric ulcers, which would have occurred, even if there had been no gastrectomy.

The important corollary is that the classical gastrectomy which removes the ulcer-prone area will prevent such ulcers. The same cannot be said for vagotomy with preservation of the ulcer-prone area.

LATE RESULTS

Gastric comfort cannot be measured quantitatively. Late results are, therefore, unsuitable for mathematical or statistical expression. The great majority of patients express unprompted their delight with the results. But there are others. My patients are asked to report twelve months after their operation. The patient thus returns, not because of symptoms, but because requested. When asked to tell the worst, some complain of occasional nausea following dietary indiscretion, febrile attacks, nervous upsets, or sometimes first thing in the morning, perhaps, even with vomiting. They report this has gone on "ever since the operation". It is apparently a troublesome sequel of the surgeon's work, and perhaps something should be done about it. They could perhaps be classed as "bad results", or "post-gastrectomy syndrome". But we turn to the very detailed pre-operative notes, and read them aloud to the patient. "Severe pain no longer relieved by powders, present for years, waking the patient at night, nausea always. Bouts of vomiting, every day in the bad spells—small quantities of food or slimy-yellow fluid—relieves the pain." Vomiting was a friend in those days. The description of these pre-operative symptoms has a peculiar force, for it is couched very largely in the patient's own words. His estimate of his operation now changes. Perhaps the result is not bad after all. He had an irritable stomach, even before his operation. At this stage, he gives a very emphatic "No", when asked if he would like his operation undone. I, therefore, conclude that his "syndrome" has a better title to be grouped under "pre-gastrectomy" than under "post-gastrectomy".

Then there are two or three patients whose symptoms remain as they were before the operation. At operation, the "visible palpable lesion" was not found. Operation was done in the belief that the symptoms may have depended on a marked antrum-gastritis. But the microscope revealed only minimal and superficial gastritic changes. These were failures of diagnosis.

No patient has been made worse by operation, nor has capacity for work been diminished. On the contrary, all those who had given up work on account of their ulcer have resumed their original work after operation. Farmers are again enabled to ride their horse, grub-out stumps from their paddocks, and shift their 180 lb. bags of wheat. There are no post-gastrectomy "cripples" attending the gastro-enterological Clinic at St. Vincent's Hospital. Therefore, when colleagues from other hospitals tell me that "We are not keen

on gastrectomy in our hospital", I know that their results have been unsatisfactory, and that they have not mastered the operation.

In my opinion, partial gastrectomy cannot be improved upon as a surgical treatment for surgical ulcer. It has reduced the indifferent and poor results to those patients whose symptoms depend upon more diffuse causes than the ulcer, and who, therefore, belong to a group which cannot be further reduced by surgery. Perhaps the medicine of the future offers hope.

CONCLUSION

Our search for an ideal has terminated, thanks to the work of many men, not least of which is that of the great Australian surgeon, whom we delight to honour in our journal. Unfortunately, the contribution which we would most gladly have seen in this number of the journal, that from his fellow-worker and fellow-author, his son, has been forestalled by untimely death. The respect and sympathy of surgeons goes out to Sir Hugh, and, in similar spirit, to the families of those other great gastric surgeons whose lives have been shadowed by tragedy — to the Judins, the Finisterers, the Eiselsbergs and the Polyas. For Judin was overtaken by that mysterious disappearance which stalks in Russia today; Finsterer lost his son, a medical practitioner, to Russian slave-labour after World War II; von Eiselsberg was killed in a railway accident caused by sabotage, in those troubled days when Hitler's gang shot Dolfuss and allowed him to bleed to death in his own chancellery, laid violent hands upon Austria, and plunged her into World War II; and Polya was murdered in Budapest by the Nazi and Communist ruffians, who pillaged the city at the end of World War II.

Thirty years ago in Vienna, I gave to von Eiselsberg the stamps from my Australian letters for his young son. Until recently that son was numbered among the Diplomatic Corps in our own Federal Capital City, and it is a great satisfaction, also, to know that Polya's son has found shelter in this country, where he enjoys in peace his home, his family and his farm.

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THE MANAGEMENT OF OVERLOOKED AND RECURRENT STONES IN THE BILE DUCT, WITH SPECIAL REFERENCE TO TRANSDUODENAL CHOLEDOCHAL SPHINCTEROTOMY

By Rodney Maingot, F.R.C.S.

London

DURING the last ten years the operation of transduodenal choledochal sphincterotomy has been performed with increasing frequency and success for patients suffering from overlooked and recurrent stones in the bile ducts, for spasm or fibrosis of the sphincter of Oddi, and also for cases of so-called recurrent pancreatitis and a number of the complications of acute and chronic pancreatitis.

This article, which deals with the management of retained or persistent stones in the bile ducts, is opportune in view of the highly satisfactory results which have followed the adoption of this operation in cases where the routine methods of exploration, with or without operative cholangiography, have proved disappointing. Following cholecystectomy combined with exploration of the bile ducts, as well as after secondary operations for retained or recurrent stones, the incidence of overlooked stones in the biliary passages remains high, as Sir Hugh Devine and many other surgeons have stressed.

It should be remembered that an overlooked, residual or retained stone is one which, having developed in the gall-bladder, has passed through the cystic duct into the common bile duct, and at operation has escaped detection; whereas recurrent stones are those which have re-formed in the ducts themselves, usually as a result of partial or intermittent obstruction of the choledochus, of spasm or fibrosis of the sphincter of Oddi, or superadded infection, and possibly of haemolytic crises.

That stones can re-form in the ducts themselves is, of course, a well-accepted fact (Fig. I). The differential diagnosis of re-formed or recurrent stones from stones which are overlooked or residual in the biliary tree is based on several pieces of evidence. Calculi are considered to be re-formed when post-operative cholangiograms, taken before the patient leaves hospital, give no evidence of stones; when the repetition of former symptoms, such as biliary colic, vomiting, chills, pyrexia and jaundice, is delayed longer than would be the case if stones were residual; when multiple recurrences necessitate repeated surgical removal of stones over a long period of time; and when the nature of the calculi removed at re-exploration is consistent with what would be expected of re-formed stones.

In cases of recurrent or re-formed stones, symptoms usually declare themselves on an average about four to six years after primary choledochostomy or secondary bile duct exploration. Such stones are multiple or single, and may be large or small, soft or mushy, usually darkly pigmented with calcium bilirubinate incrustations; whereas overlooked or residual stones are commonly firm and faceted and are lighter in colour, although there are many exceptions to these generalizations.

Incidence of Overlooked and Recurrent Stones

The incidence of overlooked stones in the biliary passages varies with the type of operation performed. Thus, after cholecystectomy alone the incidence is about 2-3 per cent.; after cholecystectomy combined with choledochostomy in the best hands it varies from 8-12 per cent. after a period of about ten years; whereas after secondary or tertiary exploration of the bile ducts the computed rate of re-formed or recurrent stones within the same period is approximately 8-10 per cent.

It should be remembered, however, that the above figures are a generally accepted rough estimate, as the incidence of stones being overlooked or being impossible to remove at operation has been variously estimated at 2-26 per cent. in recent reports. Most authorities agree that stones are computed to

be present in the bile ducts in approximately 10-15 per cent. of patients who undergo cholecystectomy, but these figures, too, have ranged from 6.2 to 26 per cent. in reports published during the last decade (McKittrick *et alii*, 1949; Glenn, 1952; Johnston *et alii*, 1954; Hicken *et alii*, 1954; Cattell, 1948; Best *et alii*, 1953; Smith *et alii*, 1959).

FIG. I. Cholangiogram taken about four years after cholecystectomy for calculous cholecystitis. Common duct not explored at the time of cholecystectomy.

At the secondary operation the small stones which are shown at the lower end of the common bile duct were removed and T-tube drainage was instituted after confirming that the ampulla of Vater was amply patent. As these stones were soft and pultaceous it was assumed that they were recurrent or re-formed stones.

In my own series of cases the bile ducts are explored in a little more than 30 per cent. of cases of calculous cholecystitis, and calculi are discovered in rather less than one-third of these cases (Maingot, 1958).

CAUSATIVE FACTORS

There are many factors which account for overlooked or recurrent stones, and among these the following should be mentioned: poor anaesthesia, inadequate incision, poor illumination and retraction, the misinterpretation of cholangiograms, neglect of the generally accepted indications for exploration of the choledochus, haemorrhage during the conduct of operation, low physical state of the patient, acute cholecystitis associated with periductal inflammatory oedema, spasm or stenosis of the sphincter of Oddi, intrahepatic calculi, stones which are hidden in diverticular pouches or crypts in the lower reaches of the bile ducts, small putty-like stones which occupy the ampulla of Vater, and the surgeon's inexperience and unfamiliarity with the pattern of the bile ducts and his failure to detect ductal aberrations.

The greatest importance is attached to the taking of a careful history of the case, cholangiographic studies, liver function tests, and the acceptance of the well-recognized indications for exploration of the bile passages in the presence of chronic or acute calculous cholecystitis.

INDICATIONS FOR DUCTAL EXPLORATION

The indications for exploring the bile ducts during operations for chronic or acute cholecystitis may be listed as follows: (1) Past or present jaundice and subclinical jaundice; (2) Thickened or dilated common duct; (3) Multiple small stones in the gall-bladder, with capacious cystic duct; (4) Positive or suspicious findings on palpation; (5) Sediment in aspirated bile; (6) Subacute or chronic pancreatitis (and many cases of acute cholecystitis); (7) Biliary tract symptoms, e.g. repeated attacks of colic; (8) Post-cholecystectomy symptoms; and (9) Evidence of ductal stones on cholangiography.

It is now appreciated that many complications, e.g. common duct stones, acute cholecystitis, pancreatitis, etc., which result from delay in treatment of patients with gall-stones, can be avoided by early recognition and early surgical intervention. Once a diagnosis of gall-stones is established, surgical treatment should, in the majority of cases, be advised and be instituted as soon as possible.

The contra-indications to operation for calculous cholecystitis are the presence of some grave medical disorder or other more serious organic disease, and in aged and decrepit patients. It should be remembered that the

older the patient and the longer the history, the greater are the chances of complications. Likewise, the operative mortality and the post-operative morbidity in old age are more than trebled when compared with that in the younger age group.

FIG. II. Control cholangiogram, i.e. picture taken during operation but before the abdomen is closed. This skiagram shows the short T-tube in position, multiple stones in the common bile duct, and a crescentic defect at the lower end of the bile duct caused by an impacted stone in the ampulla of Vater.

The T-tube was removed and the stones in the common duct were extracted with Desjardins forceps, but the one impacted at the lower end of the bile duct could not be forced downward into the duodenum or upward into the choledochus.

A transduodenal sphincterotomy was performed, as illustrated in Fig. III.

The indications for surgical intervention are just as cogent in the case of the solitary gall-stone as when multiple stones are present. Admittedly silent stones do exist, not only in the gall-bladder but in the biliary passages; but it is impossible to concede that gall-stones are harmless because they are symptomless. Sooner or later complications ensue, and it is these complications, such as jaundice, cholangitis, pancreatitis and so forth, which increase the operative death-rate.

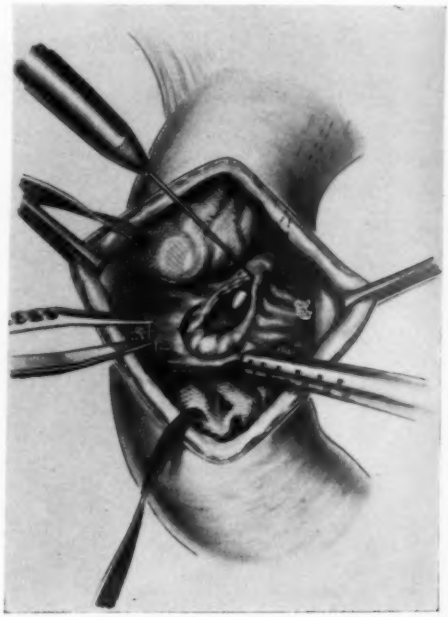

FIG. III. Sphincterotomy for impacted stone in the ampulla of Vater. Note that in this case a diathermy needle was used to divide the sphincter and to liberate the stone.

INDICATIONS FOR SPHINCTEROTOMY IN CASES OF CHOLEDOCHOLITHIASIS

(1) In those cases which have had previous operations on the bile ducts and in which residual or recurrent stones are found to be present.

(2) In all cases where it is found impossible to pass a sound, such as a small Bakes dilator, through a choledochotomy incision into the duodenum.

(3) Where Bakes dilators up to 5 mm. can be passed into the duodenum, but with difficulty.

(4) Where an impacted stone can be felt in the ampullary region but cannot be dislodged through a choledochotomy incision.

(5) Where gall-stones are associated with pancreatitis and in all cases where the ducts harbour a large number of stones.

(6) Where, following operative or control cholangiography, a stone or stones are shown to be present in the duct or impacted in the ampulla (Figs. II and III).

(7) Most cases of subacute, recurrent or chronic pancreatitis.

(8) In the majority of cases of complications of pancreatitis, such as pseudo-cyst, pancreatic fistula, etc.

(9) In cases of fibrosis of the sphincter of Oddi.

PRE-OPERATIVE EVALUATION

Most patients who are operated upon for cholelithiasis and/or for choledocholithiasis will have had oral and intravenous dye tests (Telepaque and Biligrafin) in addition to X-ray examinations of the stomach, duodenum and colon.

With complete examination of the blood, tests of hepatic function and examination of the urine and faeces, combined with these radiological studies, a high degree of diagnostic accuracy can be achieved, not merely in recognizing gall-stones and stones in the biliary ducts, but also in predicting the presence of associated gastro-intestinal or hepatic disease.

Most patients under the age of 50 with gall-stones or stones in the biliary passages, or both, but who are not suffering from jaundice, require no particular medical preparation for operation. Special care and attention are, however, needed for the aged or jaundiced patient, for the obese patient, and for those with demonstrable cardiovascular or renal disease, liver dysfunction or diabetes mellitus. A few days spent in careful preparation of these patients for elective surgery may prove decisive in the post-operative period. Blood transfusions may be indicated in the poor-risk patient, but these should be given intermittently in small amounts rather than by massive infusion. Vitamin K should be prescribed in the pre-operative period to reduce bleeding tendencies, while intravenous injections of Parentrovite will supply the patient with an ample store of vitamin B complex and ascorbic acid.

A patient who is suffering from bile duct obstruction due to an impacted calculus should not be subjected to a lengthy pre-operative regime. A few days will suffice to get him fit for surgery. Further delay might increase the damage to the parenchymal cells of the liver and kidneys.

FIG. IV. Incisions employed in biliary tract surgery:—
(a) Vertical rectus muscle-split incision;
(b) Paramedian;
(c) Subcostal or Kocher incision.

THE TECHNIQUE OF SECONDARY EXPLORATION OF THE BILE DUCTS COMBINED WITH TRANSDUODENAL CHOLEDOCHAL SPHINCTEROTOMY

The anaesthetic of choice is the sequence thiopentone-gas-oxygen-muscle relaxant. This ensures excellent relaxation and a quiet abdominal field.

The abdominal incision should be long, and a vertical one is preferable to a subcostal Kocher incision. Vertical scars which have been placed in the midline or in the inner border of the right rectus muscle should be completely excised, and the incision should then be carried down to a distance of at least three inches to the right of and below the

umbilicus. A right subcostal incision does not afford such satisfactory exposure as a vertical incision, and the chances of ventral hernia are greater after the employment of the former (Fig. IV).

After excision of the scar or scars, the rectus muscle should be divided longitudinally from the xiphisternum or right costal margin for a distance of three inches below and to the right of the umbilicus. after which the peritoneum is picked up at the lower end of the incision and incised between elevated artery forceps.

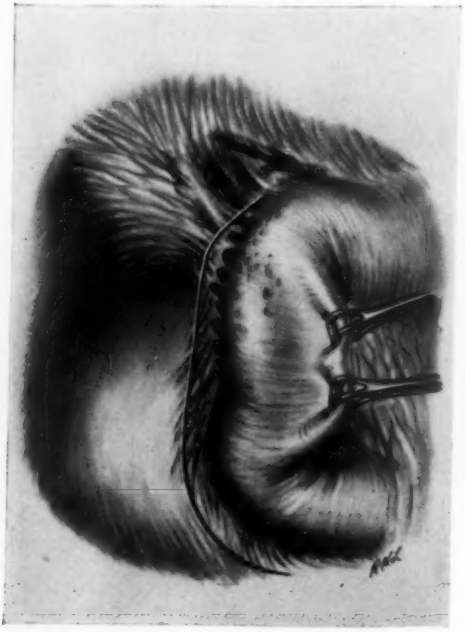

FIG. V. Secondary exploration of the bile passages. The incision used for mobilization of the duodenum.

Adhesions, whether these be omental or visceral, should be carefully freed from the abdominal wall and from the liver and right paracolic gutter, after which the ascending colon and hepatic flexure are mobilized and drawn downward and inward away from the field of operation. The stomach and duodenum should now be liberated from their attachment to the liver and the portal fissure. When this difficult liberation has been achieved, the first, second and third parts of the duodenum and the head of the pancreas are mobilized, as is well illustrated in Figs. V and IV.

The common bile duct will now come well into view, and the so-called common duct gland will be identified lying just above the head of the pancreas and close to the inferior margin of the choledochus. At this stage it is advisable to do a complete exploration of all the abdominal viscera, special attention being paid to the presence or absence of a hiatus hernia or any organic lesion of the stomach or duodenum, and to the pancreas to

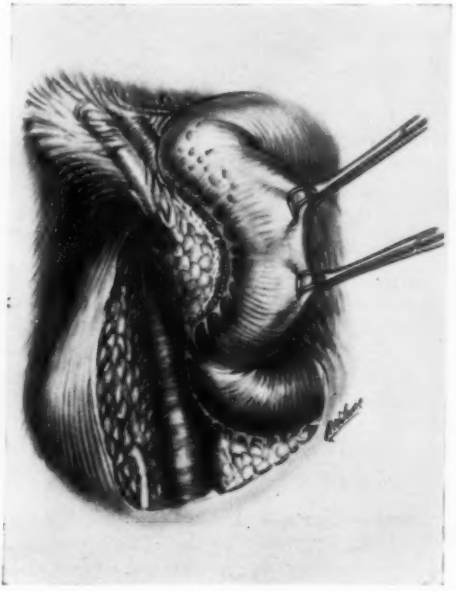

FIG. VI. Secondary exploration of the bile passages. Note the exposure gained by liberally mobilizing the duodenum and the pancreas. Note also the position of the common duct gland.

detect or to exclude the presence of pancreatitis. After palpating both lobes of the liver and the spleen, the remaining organs, such as the small intestine, colon and pelvic viscera, should be examined for any evidence of a destructive lesion. If there is no concomitant abdominal disease, the common duct should be carefully examined by sight and touch, and if there is a cystic duct remnant (pseudo-gall-bladder) this should be excised.

After packing off the operative field with a long broad roll of gauze, and after inserting

three Deaver retractors, one to draw the duodenum firmly downward so as to put the common duct on the stretch, the second to retract the right lobe of the liver upward and outward, and the third to keep the stomach from intruding into the operative field, two stay sutures are inserted in the anterior wall of the common bile duct, just below the site where the cystic duct had joined the common ducts. These sutures should be placed close to one another to allow sufficient room for an incision to be made with a knife through the anterior wall of the duct. The issuing bile is removed by suction, the small incision in the anterior wall of the duct is enlarged by opening the points of scissors, stones in the duct are removed through the choledochotomy incision with the aid of forceps, scoops, Desjardins forceps, etc., and, where possible, graduated Bakes dilators are passed into the duodenum through the choledochotomy incision.

The next stage consists in making a small transverse incision in the duodenum at the point where the Bakes dilator can be felt through the bowel wall. By elevating the olive-shaped end of the Bakes dilator, the papilla of Vater can be made to project against the anterior wall of the duodenum, thereby indicating where the transverse incision in the bowel should be made. The papilla is identified and the ampulla is dilated with small probes or mosquito forceps before introducing the dilator into the duodenum. The dilator is now withdrawn and Desjardins forceps are passed through the choledochotomy incision, down the duct through the ampulla into the duodenum. The blades are then separated to allow sufficient room for the introduction of a pair of pointed scissors. The inferior blade of the scissors is introduced into the ampulla in an outward and upward direction (11 o'clock) for a distance of 10 mm., and the sphincter is divided, as depicted in Figs. VII and VIII.

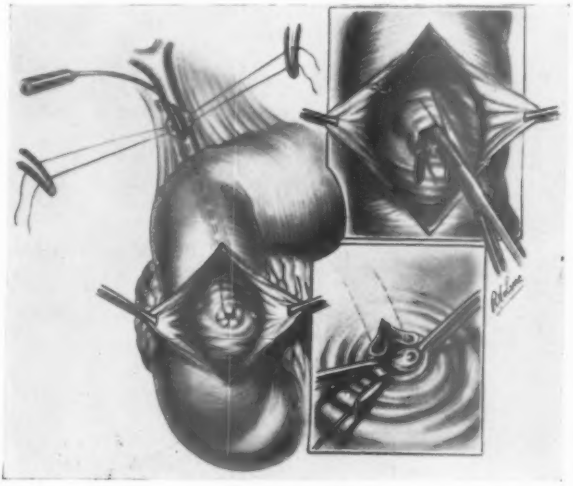

FIG. VII. Author's method of performing transduodenal choledochal sphincterotomy after exploration of the common bile duct.

It should be remembered that in many cases where stones are lodged in diverticula in the lower end of the common bile duct, or where they are soft and pultaceous, the Bakes dilators may pass freely through the ampulla into the duodenum. In other words, the surgeon may easily be misguided by this fact into thinking that there are no stones present.

Any remaining stones are milked down into the duodenum, and after thoroughly irrigating the ducts with warm saline solution, a short or long type Cattell T-tube is employed for drainage of the biliary system.

There is no need to suture the cut edges of the sphincter, as bleeding is minimal, and in

cases of overlooked or recurrent stones there is no necessity to carry out an intubation of the duct of Wirsung, as would be indicated in cases of pancreatitis.

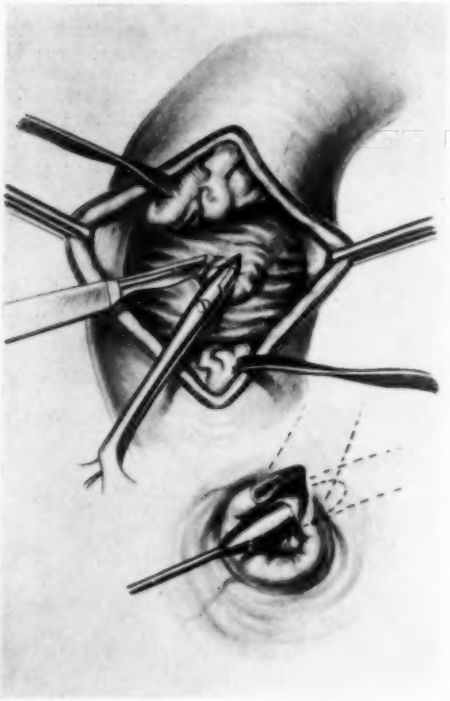

FIG. VIII. Transduodenal choledochal sphincterotomy as practised by many surgeons including Cattell.
In the inset the duct of Wirsung is being dilated with a Bakes dilator.

The surgeon should bear in mind that the ampulla of Vater lies submocosally inside the circular muscle of the duodenum, and that the incision through the papilla and ampulla upward and outward for a distance of 10 mm. will inevitably section the whole sphincter including the choledochal sphincter of Boyden. If the incision is extended higher, it may divide the circular muscle of the duodenum and destroy its "pinchcock" action which prevents duodenal reflux after sphincterotomy, as has been well illustrated by Doubilet. A long incision will also cut the artery (which we have named the circular artery) lying in the circular muscle, and produce brisk bleeding. Attempts to ligate this artery often prove exacting and may compromise the intramural portion of the common bile duct and even result in fibrous stricture.

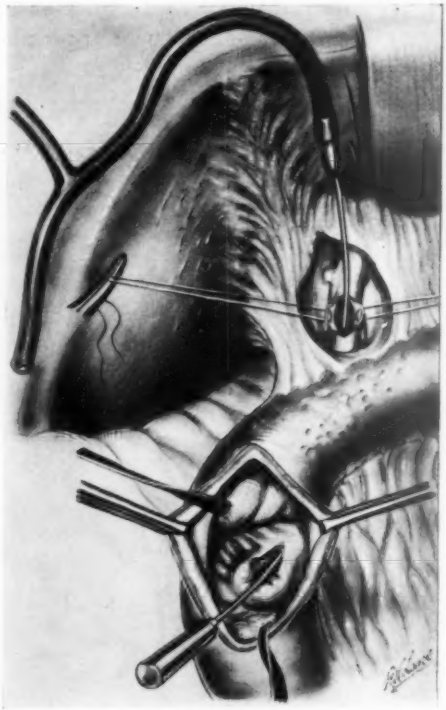

FIG. IX. Exploration of the bile passages followed by transduodenal choledochal sphincterotomy with long T-tube drainage. The figure illustrates the method of drawing the long limb of the T-tube into the duodenum.

"Again, a long incision may extend through the duodenal wall, produce a hidden retroduodenal leak, and result in abscess formation, duodenal obstruction, and even death" (Doubilet, 1958).

On the whole I prefer to use a short T-tube rather than a long one. Some authorities claim that the long descending arm of the T-tube may compress the duct of Wirsung and thus incite an attack of pancreatitis; but this has not been my experience, provided always that the size of the tube is smaller than the calibre of the duct (Figs. IX, X and XI). With the short T-tube in place, the incision around the issuing limb is sutured with a continuous suture of fine medium chromic

catgut, after which control cholangiograms are taken, employing diotrast or visciosol, to make doubly sure that no stones have been overlooked (Fig. XII).

missed, they have every chance of passing through the sectioned sphincter in the post-operative stage. Doubilet states that in cases of recurrence of multiple stones there is

FIG. X. Transduodenal choledochal sphincterotomy with T-tube drainage. The T-tube is in position, and the small choledochotomy incision is closed around the issuing limb of the tube. Note especially that the calibre of the T-tube is smaller than the opening made in the ampulla, thereby mitigating the likelihood of pancreatitis.

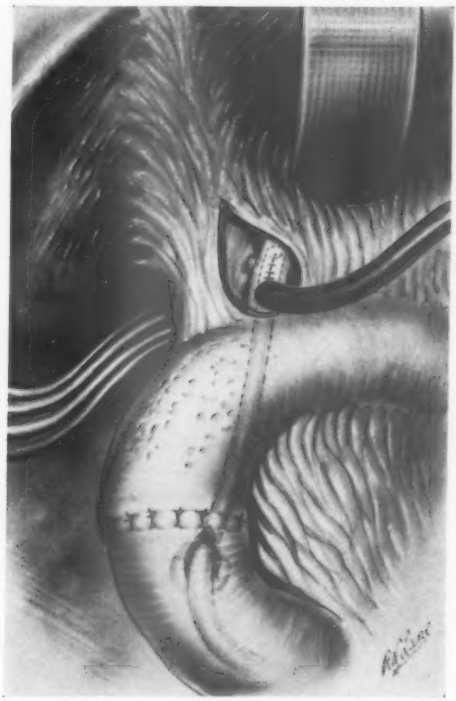

FIG. XI. Exploration of the bile ducts combined with transduodenal sphincterotomy. The operation is completed. Note the transverse closure of the opening in the duodenum and drainage of the right subhepatic space.

If a stone or stones are found on control cholangiography, these should be immediately removed and another cholangiogram be taken to ensure that the ducts are now clear. If, however, all is well, the duodenal incision is approximated transversely with a continuous suture of chromic catgut and reinforced with interrupted Lembert sutures of fine black silk, after which the abdominal incision is closed.

In my opinion, in the presence of multiple small stones in the common duct, exploration can never be complete unless the sphincter of Oddi is sectioned transduodenally and the duct explored from below as well as from above. Even if several small stones are

evidence that precipitation of bilirubin has occurred as a result of chemical inflammation of the bile duct due to reflux of pancreatic juice. Experience confirms that sphincterotomy prevents the further formation of stone by diminishing stasis and by abolishing reflux of pancreatic juice.

The operative field is always drained with corrugated rubber tubing, after which the abdominal incision is closed with a continuous lockstitch of No. 1 medium chromic catgut for the peritoneum and posterior sheath of the rectus muscle, and with interrupted nylon, silk or wire sutures for the anterior sheath of the rectus muscle. For approximating the skin edges interrupted vertical mattress sutures of fine Deknatel are routinely used.

POST-OPERATIVE CARE

Where a short T-tube has been used for drainage purposes, cholangiograms are taken on or about the 8th post-operative day. Where a long T-tube has been employed, these pictures are on the whole unsatisfactory, as most of the dye is injected down the tube straight into the duodenum. In these latter cases it is well, therefore, to tilt the patient head downward in taking these pictures, as the intrahepatic radicles are clearly outlined and the opaque medium very often seeps downward into the duodenum around the entire intraductal circumference of the tube.

FIG. XII. Short T-tube drainage following sphincterotomy. The operation is nearly completed, as the short T-tube is being inserted into the choledochus, and the small incision will be sutured around the issuing limb of the T-tube.

In the inset, various types of T-tube in common use are depicted.

If a retained stone or stones are demonstrated following post-operative cholangiography, provided the stones are small they may be left in situ in the hope that they will subsequently pass through the widened sphincteric orifice. If, however, the stone or stones in the choledochus are sizeable, have floated upward into the right or left hepatic duct, or appear to be impacted in the ampullary region, a further operation will be called for for their removal during the convalescent period (Figs. XIII, XIV and XV).

FIG. XIII. Post-operative cholangiogram taken ten days after cholecystectomy plus choledochostomy for calculous cholecystitis. The picture shows a stone retained in the lower end of the common duct with some filling of the pancreatic duct.

At secondary exploration of the duct some three weeks after the cholecystectomy, the stone was removed and the ampulla was readily dilated with Bakes dilators, the largest used being 10 mm. Sphincterotomy was not performed in this case.

I am definitely opposed to any attempt being made at dissolving or at aiding the passage of a retained stone or stones into the duodenum by the use of chemical agents or oily substances which are considered by some to be helpful for this purpose. I have seen cases in which oily solutions, such as medicinal paraffin, either singly or in combination with chloroform or ether, have been injected down the T-tube to facilitate the dissolution

or expulsion of residual stones in the ducts. This method is extremely painful, ineffective and dangerous. It may cause an acute cholangitis, acute pancreatitis or subsequent fibrosis of the duct, while the chances of success depend in large measure upon the degree of patience exerted by the user and also upon the fortitude of the patient in his desire to avoid another operation (Smith *et alii*, 1957).

FIG. XIV. Control cholangiogram following cholecystectomy for gall-stones and T-tube choledochostomy. The opaque medium has revealed at least two stones in the left hepatic duct. The left hepatic duct was immediately re explored and four stones were removed, after which another cholangiogram was carried out.

When long T-tubes are employed following sphincterotomy, it is advisable to allow the issuing limb to drain continuously into a bottle beside the bed. Under no condition should this tube be clamped off during any period of convalescence, as regurgitation of duodenal contents is very prone to take place, and such irritating juices may initiate a chemical cholangitis of the intrahepatic radicles of the bile ducts. These long T-tubes are best removed between the 10th and 15th post-operative days.

When a short T-tube is used, the issuing limb may be clamped after the first week for periods varying from six to twelve hours. Should there be no leakage of bile, no backache or other untoward symptoms, the tube may be clamped off for longer periods. Post-operative cholangiograms are usually obtained sometime between the eighth and twelfth post-operative days, and if these show that the biliary passages are clear and there is a free and unhindered flow of the opaque medium into the duodenum, the T-tube may be safely withdrawn.

FIG. XV. The same case as in Fig. XIV, showing the left hepatic duct now clear of stones, and free entry of opaque medium into the duodenum. Following this second cholangiogram on the table, the abdomen was closed and the patient made a good recovery.

CONCLUSIONS

1. The incidence of overlooked or recurrent stones in the bile ducts is at present much too high, being approximately 2-3 per cent. following cholecystectomy alone for calculous cholecystitis, 8-12 per cent. following cholecystectomy combined with choledochotomy, and approximately 10 per cent. for cases of re-formed or recurrent stones in the biliary passages following secondary exploration.
2. Whilst cholangiography undoubtedly plays a part in the detection of calculi in the choledochus, and has added much to

our knowledge of the intrahepatic ducts as well as concerning the functional and pathological abnormalities of the biliary tree, in my opinion this method must become more accurate and dependable before it can replace surgical exploration of the ducts by experienced surgeons or become a routine part of choledocholithotomy.

3. Whenever the biliary passages are explored for stone, a control cholangiogram (that is, one taken before the abdominal incision is closed) may with advantage be carried out to provide supportive evidence that the ducts are clear.

4. When the ducts are explored, whether stones are found or not, T-tube drainage should always be instituted to afford means of carrying out post-operative cholangiography.

5. The indications and contra-indications of sphincterotomy have been outlined, and the technique and the precautions necessary when undertaking this procedure have been considered in some detail.

ACKNOWLEDGEMENTS

I wish to acknowledge with grateful thanks the drawings of bile duct surgery for overlooked and recurrent stones, which were done by Mr. Robert Lane at operations performed by me at the Royal Free Hospital, London, and at the London Clinic. Mr. Lane has taken a great deal of trouble in reproducing in some detail and with great accuracy these operative procedures.

I am also indebted to the following colleagues who are consultant radiologists to various hospitals in Great Britain, namely, Dr. D. C. Staveley and Dr. E. B. Madden, of the Royal Free Hospital, London; Dr. C. G. Whiteside, of the Middlesex Hospital, London; Dr. David Stenhouse, of the Western Infirmary, Glasgow; and Dr. D. K. L. Davies, of St. David's Hospital, Cardiff.

These consultant radiologists have taken considerable pains in collecting skiagrams to illuminate this article and also to emphasise the part that radiology plays in the diagnosis, and in fact in the treatment, of retained calculi in the biliary passages.

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INVESTIGATION AND PREPARATION OF THE JAUNDICED PATIENT PRIOR TO OPERATION*

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WHEN a patient suffering from jaundice presents for treatment, it is imperative that every endeavour should be made to achieve an accurate diagnosis — to determine the cause of the jaundice, be it gall-stones, infectious hepatitis or cancer. Secondly associated conditions should be detected such as metabolic diseases including diabetes, infections such as pneumonia or pyelonephritis, or malnutrition from alcoholism. It is often wise for both physician and surgeon to work together from the outset. Clinical observation, radiological investigation and biochemical tests are the sheet-anchor of diagnosis. On occasions liver biopsy is indicated: this may reveal the correct diagnosis and so prevent an unnecessary laparotomy, or may call for a laparotomy where "hepatitis" has been diagnosed in error.

If surgery be indicated, all resources should be brought to bear to produce maximal clinical improvement before operation. Firstly, an attack should be made on the underlying causes of the illness by medical means. Thus antibiotics such as penicillin and streptomycin may be injected, or perhaps broad spectrum antibiotics of the tetracycline group may be given by mouth to counter cholangitis (Sherlock, 1956; Twiss *et alii*, 1956). Secondly, any associated disease, such as diabetes, renal infection or malnutrition, may require urgent medical treatment. Thirdly, jaundice may not only be accompanied by failing liver function, but frequently it may be associated with profound anorexia, vomiting and perhaps anaemia from blood loss which may be related to hypoprothrombinaemia. This calls for replacement therapy by the intravenous infusion of blood or serum, water, glucose, vitamins particularly vitamin K, and electrolytes, particularly sodium chloride and perhaps potassium. The patient's clinical state and biochemical examinations of the blood and urine are of the greatest value in planning replacement and maintenance therapy. Where possible a high carbohydrate diet is given. It is unwise to force the proteins if there be liver failure. Sedatives should be given with caution, as jaundiced patients with impaired liver function may show a pronounced response to the standard dosage.

Anaesthesia

Finally, the anaesthetist should be brought into the picture in adequate time to enable him to assess the clinical status of the patient. It is essential that he should provide optimum anaesthesia for a maximum time. Operations on the biliary tract are most exacting and time consuming. If the surgeon is hastened in his endeavours by a failing in the patient's clinical condition, then imperfect surgery may result. Imperfect surgery is denoted by inaccurate diagnosis at operation and incomplete surgical cure or relief of the jaundice.

Impairment of liver function in the jaundiced patient presents added hazards to the anaesthetist, and we are indebted to Dr. Patricia Wilson for the following comments. Firstly, hypoxia may cause more damage to the liver than the anaesthetic agent itself, and must be prevented by maintenance of full oxygenation and normal blood pressure, and adequate blood replacement. Secondly, anaesthetic agents which may cause liver damage

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are best avoided, particularly chloroform, ether, trichlorethylene and halothane. Safer agents are cyclopropane and nitrous oxide (Bourne and Rosenthal, 1928; Bourne and Raginsky, 1934; Armstrong, 1947). Thirdly, drugs which are poorly detoxified by the damaged liver should be used with great caution: these include morphine, pethidine, and barbiturates such as thiopentone (Dundee, 1955). Also the muscle relaxants gallamine triethiodide (Flaxedil) and d-tubocurarine (Tubarin) are partly metabolized by the liver (Everett, 1948); when liver function is impaired the safest relaxant is a suxamenthonium compound such as succinyl choline (Scoline).

CLINICAL EXPERIENCE

In the light of this plan of diagnosis and management, what has been our experience? What has been the cause of jaundice? How have the diagnoses been made before and during the operation? How accurate have been these findings? What has been the mortality in the various diseases causing the jaundice, treated with or without surgery?

To answer these questions our experience during the past ten years in the Clinical Research Unit of the Royal Melbourne Hospital and the Walter and Eliza Hall Institute was examined by selecting at random 100 cases of jaundice. Pronounced clinical jaundice (serum bilirubin over 2.0 mg. per cent.) was the sole factor in rendering them eligible for selection.

Causes of jaundice

Fig. I. shows the cause of jaundice in these 100 cases. The commonest cause was gallstones (36 per cent.) followed by hepatitis (16 per cent.) and carcinoma of the pancreas (9 per cent.). These were followed by a number of causes, all less than 10 per cent. and including cancer of the bile duct, stomach and gall bladder, nutritional cirrhosis due to alcoholism, chronic pancreatitis, hydatid disease, lupoid hepatitis, chlorpromazine jaundice and primary biliary cirrhosis.

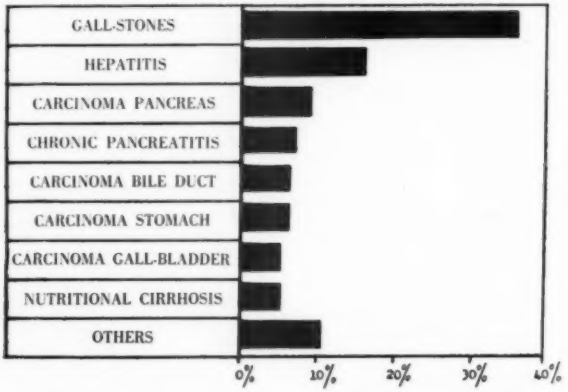

FIG. I. Causes of jaundice in 100 cases selected at random. Gall-stones and hepatitis were the most frequent causes.

The diagnosis

Clinical observation.

The diagnosis was made firstly by clinical observation including the presence of spider naevi, ascites, enlargement of the liver, gallbladder and spleen, and the presence of xanthomatous deposits. Daily scrutiny of the stools was most helpful.

Biochemical tests.

These were of considerable value in determining the cause of the jaundice. Estimations were made of serum bilirubin (Malloy and Evelyn, 1937), cephalin flocculation (Hanger, 1939), fractionation of the serum proteins (Hawk *et alii*, 1939; Gornall *et alii*, 1949), γ-globulin by the zinc turbidity method (Kunkel *et alii*, 1948), and alkaline phosphatase (King and Armstrong, 1934). The results in the 100 patients are given in Fig. II. A negative cephalin flocculation test and a high alkaline phosphatase were the most informative tests in deciding whether there was obstructive jaundice requiring surgery. This

has been the experience of others (Hanger, 1954). In those instances where a positive cephalin flocculation was obtained in conjunction with a high alkaline phosphatase, normal values for the serum proteins, especially γ-globulin (Wilson *et alii*, 1957), favoured obstructive jaundice. The prothrombin levels (Quick, 1935) were frequently low, particularly in cases of chronic hepatitis.

It is of interest to note that all 5 cases of chlorpromazine hepatitis in this second series gave the biochemical findings of obstructive jaundice (Fig. III). Moreover the SGO-T levels were not characteristic (Fig. III). Chlorpromazine hepatitis thus mimicked conditions demanding of surgery—a warning to the surgeon.

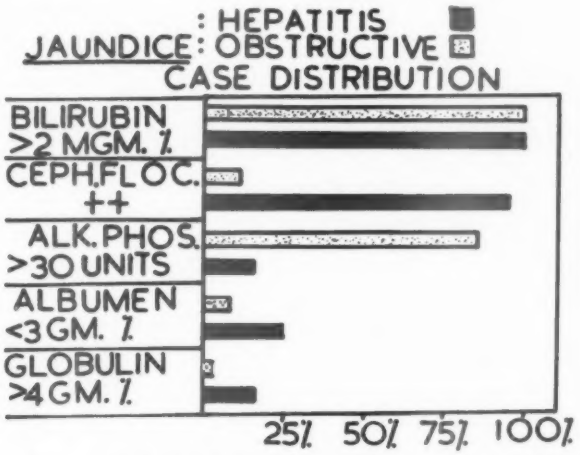

FIG. II. Biochemical findings in the 100 cases of jaundice. A negative cephalin flocculation test and a pronounced rise in alkaline phosphatase were most valuable in indicating jaundice requiring surgery.

Recently, the determination of serum glutamic-oxalacetic transaminase (SGO-T) (Reitman and Frankel, 1957, modified by Holden and Weiden, 1958) was introduced to our Unit. In view of this a second series (Fig. III) of 50 cases of jaundice recently studied has been analysed with reference to the SGO-T and γ-globulin levels in addition to the tests used in the main series (*vide supra*). As in the main series, the cephalin flocculation test and alkaline phosphatase level were the most helpful; also the presence of a low albumen level and particularly a high γ-globulin level (Fig. III) favoured a diagnosis of hepatitis. The SGO-T level was estimated in 26 of this second series including 14 patients with hepatitis, 7 with obstructive jaundice and 5 with jaundice due to chlorpromazine. All, except 5 with hepatitis, gave values over 25 units (normal range 15-25 units). In our experience this test thus proved to be of no value in differential diagnosis, but it was a measure of liver involvement (O'Brien *et alii*, 1958).

Hydatid tests.

The Casoni skin test and hydatid complement fixation test were performed in cases suspected of hydatid infection. However, the Casoni test is of limited value in the presence of jaundice. Only one case of hydatid disease was detected in the series.

Blood culture.

In 2 cases of jaundice a positive culture was obtained (*E. coli* and *S. pyogenes*), the illness being accompanied by shivers, pyrexia and leucocytosis. In such cases the nature of the organism and its sensitivity to antibiotics can be a valuable guide to treatment.

Liver biopsy.

Today the greatest call for liver biopsy, whether or not jaundice is present, is to establish the diagnosis of parenchymatous liver disease, including nutritional cirrhosis, chronic hepatitis, haemochromatosis, secondary carcinoma and the multiple granulomata of tuberculosis, sarcoidosis or brucellosis.

Percutaneous needle biopsy was performed in 28 of the 100 cases of jaundice in the main series. Most of these biopsies were carried out in the earlier period of the review, for with increasing experience the distinction between "medical" and "surgical" jaundice could be made with considerable accuracy on the clinical and biochemical findings alone, without recourse to biopsy. However, biopsy was still required on occasions, when the diagnosis remained obscure.

Thus liver biopsy proved of considerable value in diagnosis where clinical observation and biochemical tests failed to indicate the cause of jaundice, particularly with regard to the necessity for operation. As subsequently stated, one patient suffering from nutritional hepatitis with jaundice was operated upon in error, and died — liver biopsy probably would have been a life-saving measure.

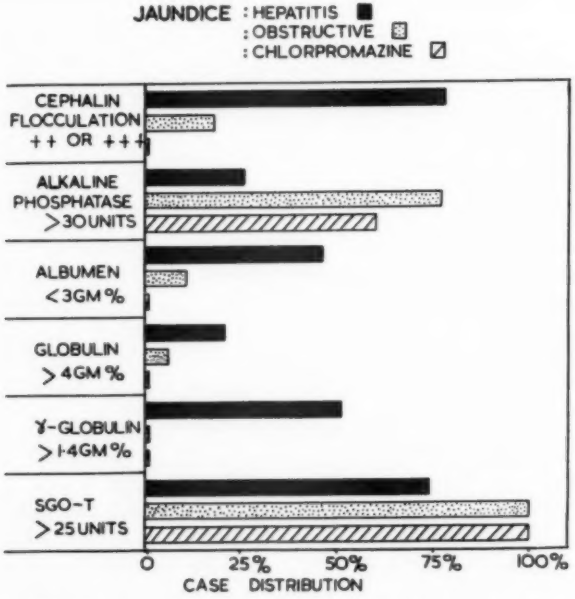

FIG. III. A second series of 50 cases of jaundice subjected to a wider biochemical survey. The cephalin flocculation test and the levels of alkaline phosphatase, γ-globulin and albumen were the most valuable tests in selecting cases for surgery. However cases of chlorpromazine jaundice gave the picture of obstructive jaundice. The transaminase test was of no value in selecting cases for operation.

Table 1 records the findings in the 28 cases subjected to liver biopsy and it will be noted that the biopsy was decisive in indicating obstructive or non-obstructive jaundice in all of the 10 cases where biochemical tests were either incorrect (3 cases) or equivocal (7 cases); moreover in 6 of these 10 cases the diagnosis by biopsy was conclusive or highly suggestive of the actual cause of jaundice (hepatitis 3, nutritional cirrhosis 1, chlorpromazine jaundice 1 and reticulosarcoma 1). In all of the remaining 18 cases the biopsy was confirmatory of the biochemical tests and final diagnosis.

Accuracy of diagnosis.

In the series of 100 cases the accuracy of diagnosis, made soon after admission of the patient to hospital and before operation, is set out in Table 2. The diagnosis was correct in a high percentage of cases of hepatitis (93 per cent.), nutritional cirrhosis (80 per cent.) and gall-stones (79 per cent.). However the precise diagnosis of the presence and site of cancer was inaccurate where cancer of the bile duct or gall-bladder was concerned. When the presence of cancer was considered irrespective of site, then the ac-

curacy of diagnosis was 90 per cent. (Table 3).

Diagnosis at laparotomy.

In some cases the final diagnosis of the cause of jaundice remained for the surgeon to make at laparotomy. This called for a well organized plan of surgical procedure including careful inspection and palpation of the liver, bile duct and pancreas. When "hepatitits" was suspected, an operative cholangiogram, performed through a fine needle in the common bile duct, soon excluded obstruction by stone or cancer of the hepatic ducts (Brown, 1959).

Liver biopsy and examination of a frozen section during the operation sometimes helped to exclude secondary cancer of the liver which might be mimicked by the hyperplastic nodules of chronic hepatitis. Occasionally biopsy of the pancreas was indicated in order to distinguish between carcinoma and chronic pancreatitis, although an absence of cancer cells does not exclude cancer.

TABLE 1

VALUE OF LIVER BIOPSY IN 28 CASES OF JAUNDICE

Final diagnosis	Accuracy of liver biopsy		Accuracy of biochemical diagnosis		
	Correct	Incorrect	Correct	Incorrect	Equivocal
OBSTRUCTIVE GROUP					
Gall-stones	7	0	4	2	1
Cancer of bile ducts or pancreas	6	0	5	0	1
Secondary carcinoma of liver	1	0	1	0	0
Reticulosarcoma of liver	1	0	0	0	1
NON-OBSTRUCTIVE GROUP					
Hepatitis	8	0	5	0	3
Nutritional hepatitis	3	0	2	0	1
Chlorpromazine hepatitis	1	0	0	1	0
Pregnancy	1	0	1	0	0
	28	0	18	3	7

Table 1—The accuracy of diagnosis was assessed in terms of the ability to determine whether the jaundice was *obstructive* and requiring surgery, or *hepatocellular* and not requiring surgery. In the obstructive group the actual cause of the obstruction was not indicated by the biopsy unless secondary cancer was present in the liver. Chlorpromazine jaundice is included in the non-obstructive group although there was gross bile plugging and the chemical tests indicated obstruction, similarly with the case of jaundice in pregnancy as described by Sherlock (1958).

Results of treatment

The results of operative and non-operative treatment in these 100 cases of jaundice selected at random are shown in Table 4. Gall-stones were treated successfully by operation in the majority of cases and there were no post-operative deaths. The immediate post-operative mortality in cancer was high, a challenge to physicians and surgeons to make an earlier diagnosis in our patients. It is comforting to observe that no case of hepatitis was subjected to laparotomy; however,

TABLE 2

JAUNDICE — ACCURACY OF DIAGNOSIS

	Number of cases	Correct diagnosis (percentage)
Hepatitis	16	93
Nutritional cirrhosis	5	80
Gall-stones	36	79
Carcinoma: Pancreas	9	77
Bile duct	6	0
Gall-bladder	5	0
Stomach	6	50
Chronic pancreatitis	7	43
Others	10	60
Total	100	

Table 2—Accuracy of diagnosis in 100 cases of jaundice selected at random. It will be noted that hepatitis, nutritional cirrhosis, gall-stones and cancer of the pancreas usually were diagnosed correctly, whereas the diagnosis of chronic pancreatitis and cancer of the bile duct, gall-bladder and stomach was inefficient.

TABLE 3

JAUNDICE — DIAGNOSIS OF "CARCINOMA" IRRESPECTIVE OF SITE

	Number Of cases	Correct diagnosis (percentage)
Carcinoma Diagnosed as being *Present*	20	90
Carcinoma Diagnosed as being *Absent*	80	90

Table 3—The diagnosis of the presence or absence of cancer, irrespective of site, was efficient.

one case of nutritional cirrhosis due to alcoholism was operated upon in error and died after operation.

Lupoid hepatitis, cholangiolitic hepatitis and primary biliary cirrhosis

Lupoid hepatitis.

The syndrome of lupoid hepatitis has been defined by Mackay *et alii* (1959) as active progressive hepatitis associated with a positive L.E. cell test, and it is thought to be perpetuated by continuing auto-destruction of liver tissue by the host's immune mechanisms.

TABLE 4

JAUNDICE — SURGERY AND MORTALITY — 100 CASES

	Survived		Died	
	Operation	No operation	Operation	No operation
Gall-stones	28	7	0	1
Carcinoma: Pancreas	4	0	5	0
Bile duct	4	0	1	1
Gall-bladder	2	0	1	2
Stomach	1	0	3	2
Chronic pancreatitis	4	2	0	1
Hepatitis	0	16	0	0
Nutritional cirrhosis	0	4	1	0
Others	3	5	2	0
Totals	46	34	13	7

Table 4—The survival rate in 100 cases of jaundice selected at random. The recorded deaths occurred during the hospital admission concerned.

The patient in the present series exhibited the typical features of the condition including intermittent jaundice, arthritis, hepatosplenomegaly, high globulin level and a positive L.E. cell test. Liver biopsy showed extensive cell damage, lymphocytic infiltration and fibrosis. There was a positive result to the auto-immune complement fixation (AICF) test described by Gajdusek (1957) and further developed by Mackay and Gajdusek (1958) of our Institute. She responded to cortisone therapy as shown by the subsidence of the jaundice and fall in bilirubin and gamma globulin, and the rise in albumin. However a more dramatic index was the abrupt fall in transaminase level (Case 1 of O'Brien *et alii*, 1958). She has remained well on continuing cortisone treatment.

Cholangiolitic hepatitis and chlorpromazine jaundice.

The series of 100 cases included 2 patients with cholangiolitis, the aetiology being viral hepatitis (1 case) and chlorpromazine toxicity (1 case). Cholangiolitic hepatitis is a viral hepatitis wherein the maximum damage appears to fall on the cholangioles rather than the parenchymal cells of the liver. Jaundice may persist for many weeks after the initial attack and the liver function tests indicate obstructive jaundice. Liver biopsy shows extensive plugging of the bile capillaries with bile thrombi; this is best seen on frozen section of the biopsy fragment, for much of the bile pigment is dissolved out during the preparation of a paraffin section. Steroids and ACTH may be of value both in diagnosis and treatment of cholangiolitic hepatitis (Shaldon and Sherlock, 1957; Summerskill and Jones, 1958). Solem (1957) found that in cholangiolitic hepatitis there was a prompt fall in the bilirubin level following administration of ACTH, whereas no fall occurred in cases of obstructive jaundice. Jaundice following the administration of chlorpromazine and other drugs such as arsenic, thiouracil and testosterone may be associated with abdominal discomfort or even pain and rigors. Eosinophilia is often present and suggests an allergic basis. The liver function tests indicate obstructive jaundice (*vide supra*), the alkaline phosphatase usually being high and the cephalin flocculation negative. The liver biopsy (frozen section) shows extensive bile thrombi and usually there is only minimal inflammatory reaction in the portal tracts (Sherlock, 1958). Occasionally the jaundice may persist for three months or more (Van Ommen and Brown, 1955); this may cause considerable

concern regarding the diagnosis. Most cases recover completely, even in the few instances where the illness has been severe and prolonged.

Primary biliary cirrhosis.

Although there was only one case of primary "xanthomatous" or "non-obstructive" biliary cirrhosis in the present series, several have been seen in the Unit. The lesion is presumably at the level of the cholangioles, and the aetiology is unknown; there is evidence that an auto-immune reaction may be responsible in some cases (Mackay, 1958). Primary biliary cirrhosis usually affects females aged 35 to 50 years, and begins with gradually deepening jaundice and severe pruritus. Characteristic cutaneous xanthomatous deposits appear in the eyelids, palms and elbows. The liver function tests show biliary obstruction and the serum lipid and cholesterol levels are highly elevated. Liver biopsy reveals a biliary type of cirrhosis with cellular infiltrations in the portal tracts. Laparotomy must be undertaken to exclude extrahepatic obstruction; this is best accomplished by a carefully performed operative cholangiogram which should easily fill the entire intrahepatic biliary system; further surgery should then be avoided.

CONCLUSIONS

Accurate diagnosis of the causes of the jaundice and of any associated diseases is of the greatest value in the treatment of the jaundiced patient. It enables full medical treatment to be given before surgery is undertaken. The surgeon must ensure that the patient is as fit as possible so that he may withstand optimal operative procedures. It usually pays to wait several days while diagnostic measures and medical treatment are being carried out. Occasionally, in cases of obstructive jaundice with severe cholangitis, it will be observed that severe liver failure is threatening, and is not being relieved by medical treatment; here heroic surgery alone will save life (Mackay *et alii*, 1957). The physician must assist the surgeon by consulting him at the outset. The anaesthetist must not flinch from his responsibility.

When the operation is completed the patient will require careful and continuing observation and restoration. This period will be the final test of adequate pre-operative preparation, optimum surgery and optimum anaesthesia. Now various challenging stresses may come into the picture such as infection in the liver area and lungs, adynamic ileus, haemorrhage, bile peritonitis, and finally fluid and electrolyte disturbances made worse by both gastric suction and liberal drainage of bile from the T-tube placed in the common bile duct. The ill effects of these stresses must be recognized and countered; the advance in knowledge of their prevention and cure has been a triumph in modern surgery. It is now appreciated that the post-operative management is as important as the pre-operative management and the operative technique.

In conclusion it must be emphasized that efficiency in diagnosis and pre-operative preparation of the jaundiced patient leads to efficient operative technique, and ensures the most satisfactory treatment.

SUMMARY

1. In the jaundiced patient, all resources should be brought to bear to achieve an accurate pre-operative diagnosis and to obtain maximal clinical improvement prior to operation; an attack must be made on both the underlying illness and any associated diseases.

2. The anaesthetist will play an important role when liver function is impaired, for drugs and anaesthetic agents known to have a hepatotoxic action must be avoided.

3. A detailed analysis of 100 cases of jaundice has been made from the standpoint of causation and diagnosis. The cephalin flocculation test and the levels of serum alkaline phosphatase and gamma globulin proved most valuable in selecting cases for surgery; liver biopsy was required when the diagnosis remained obscure.

4. The pre-operative accuracy of diagnosis was high in hepatitis (93 per cent.) and gall-stones (79 per cent.). The presence of cancer was diagnosed with accuracy (90 per cent.), but diagnosis of the primary site was inaccurate. The chemical tests in chlorpromazine jaundice suggested obstruction.

5. The results of surgical treatment of gall-stones were most satisfactory; the treatment of cancer was unsatisfactory and calls for improved methods of diagnosis and management.

ACKNOWLEDGEMENTS

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THE PATHOLOGY AND CLASSIFICATION OF TALIPES

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Definition of term

THE word talipes is often so loosely used as to lose all meaning except that of "something wrong with the foot." If one speaks of spastic and paralytic talipes one might as well use the term for the results of accidents, or for fused, extra or missing toes. I suggest that talipes should be taken as meaning a condition in which the foot has all its normal elements present, but is congenitally mis-shapen in a way that could be reproduced by the action of external moulding forces. That is to say that it is essentially the same kind of deformity, produced before birth, that used to be produced after birth by binding the feet of high-born Chinese girls. I admit that this definition goes very close to assuming the pathology suggested in this paper, but I hope to show that, if the definition is accepted and all relevant factors taken into account, the pathology inevitably follows from an analysis of the different types and their associated conditions.

Pathology

General remarks

It is impossible to arrange the feet inside the uterus in such positions that they do not, in one way or another, come in contact with the inner wall of its cavity. Consequently in moulding deformities it is always possible to find a convex surface corresponding to the uterine concavity, and sited so that it corresponds either to abnormal position or abnormal pressure.

All of the three main groups of deforming factors described below can exist together as well as separately, e.g. it is common to find malposition combined with increased pressure. Any or all of them may also be combined with genetic failures of development such as syndactyly or polydactyly; or with those congenital constrictions which I have suggested are due to parts of the body having been thrust through the amniotic membrane.

It is probably because of the complexity of many cases into which moulding enters as one factor that its existence and importance is not generally realized.

One of the strongest confirmations of the moulding hypothesis is the occurrence of what may be called "mutual deformities." This consists in deformities of the two feet which have the relation to each other of a coin and the die which struck it; that is to say though of different shape they fit snugly together. This is especially common in the extreme degrees of pressure and malposition which are assumed to cause the syndrome of Arnold-Chiari deformity plus lumbar spina bifida plus talipes.

Types of Deforming Forces

1. *Malposition*

The normal position of the lower limbs in foetal life is with the thighs flexed on the body, the knees bent, and the soles of the feet pressed against the uterine wall. Owing to this position the range of movement of the feet of the new-born baby is different from that of the adult in having a much greater range of movement into calcaneus, and a consequently decreased range into equinus. The excess of power in the calf muscles over those that dorsiflex the foot soon changes this after birth. Any departure from this position inevitably brings new forces to bear on the feet, with pathological results of varying degree.

It is curious how little attention has been given to the question of foetal position. I have not been able to find any definite pronouncements on the subject in textbooks of embryology, and works on obstetrics show figures which do not correspond to specimens of actual full-time pregnancies either in position of the feet or in tightness of packing of the foetus.

2. *Increased mechanical pressure*

As I have said, it is difficult to find authentic information as to the degree of tightness which which the foetus is held in its cramped quarters. My own impression is that the

pictures in textbooks of obstetrics exaggerate considerably the clearance between the foetus and the uterine walls. Exact measurement of the amount of amniotic fluid, and comparison of this with the clearance it would give to a folded foetus inside a suitable container should give useful information. It would obviously take a large amount of fluid to increase the clearance by even a couple of millimetres.

FIG. I. Double metatarsal varus talipes, held in position assumed to have caused deformity. Note that the pressure of the uterine wall has bent the little toes round the dorsum of the foot, and caused "compression dimples" on them.

If this suggested increase of pressure or (what is the same thing) decrease of space round the foetus did occur it would have two effects, owing to the interference with circulation that it would cause, particularly to the venous return. These are firstly defective development or atrophy of the muscles, and secondly stiffening of the joints from periarticular infiltration. Such abnormalities, of course, form the condition of arthrogryposis; and it is part of the argument that in mechanical pressure they are confined to the exposed legs and feet, the arms and hands being sheltered by the overhanging foetal head. Naturally, the stronger the pressure, the more marked both the deformity and the arthrogrypotic condition of the tissues.

In addition to the deformity as found at birth there is another effect of compression in what may be called "compression dysplasia." This is a failure of those parts which have taken the greatest compression stress to grow during development as well as those which have escaped comparatively lightly. This is particularly marked on the inner side of club feet and accounts for the notorious tendency of that deformity to recur, in spite of complete over-correction.

FIG. II. Double metatarsal varus, completely untreated, at age of five years. Note that though the feet are ugly, there is no crippling and the muscles of calf are normal.

3. *Increased hydraulic pressure*

Has effects much like those of increased mechanical pressure, but by the laws of hydraulics these are equally distributed throughout the system involved. Thus while increased mechanical pressure, as has been said, mainly affects the exposed legs and feet, increased hydraulic pressure affects all four limbs equally. The effect is a mechanical loading of the circulation, particularly the venous return, so that atrophy of the muscles and stiffening of the joints occurs, increasing in severity with the distance from the impelling pump of the maternal heart.

Although in this condition the clearance between the foetus and the uterine walls is usually (though not invariably) greater than normal owing to hydramnios, yet moulding forces come powerfully into play as the rigid

joints hold the defective tissues of hands and feet firmly against the surrounding concavity, with consequent fixed convexities on the appropriate surfaces (Fig. VIII).

FIG. III. Right metatarsal valgus talipes, and left normal foot. There was a congenital dislocation of the hip on the right side.

REGULAR FORMS OF TALIPES

1. *Metatarsal varus*

This is an effect of malposition, consisting in the soles of the feet being placed against each other, instead of pressing against the inner surface of the uterus. The consequence is that the fore parts of the feet are bent inwards to correspond with that curve. The heel and ankle are unaffected and there is no interference with nerves or blood vessels; in consequence the muscles are strong and the joints supple. Because of the pressure on the outer sides of the feet, the knees are swung outwards so that the thighs are in abduction with a corresponding abnormal range of movement in that direction after birth. Abduction of the hips prevents dislocation, and accordingly in a very large experience I have never seen congenital dislocation of the hip with a metatarsal varus of the foot on that side. This deformity is constantly either overlooked, being confused with the common inward clutching of the feet of the new-born; or else mistaken for the much more serious condition of true club foot. It is the easiest of all deformities of the feet to cure if taken early.

2. *Metatarsal valgus*

This is an effect of increased mechanical pressure, or tightness in utero. It consists in an exaggeration of the normal calcaneus position of the new-born foot which is, so to speak, wrapped round the outer side of the leg by pressure on the sole and so bent into an outward curve, the opposite of that of metatarsal varus. Two effects follow from the increased pressure. The first is that the leg muscles combined with feet of this kind are always weak, and secondly, that there is a well-recognised common association of these feet with congenital dislocation of the hip. The reason for this is that the same pressure that forces the foot up round the leg also tends to force the knee downwards and backwards, and so the head of the femur out of the acetabulum.

FIG. IV. Double "total varus" of club-foot talipes. Note:—
(i) That the feet are in calcaneus, and consequent inappropriateness of term "equino-varus."
(ii) That right foot is more severely deformed than left.
(iii) Points of extreme pressure, marked with a cross, causing "pressure dysplasia."
(iv) The convexity on the dorsal surfaces corresponding to the moulding concave surface of the uterus.
(v) The stretching and pressure to which the peroneal muscles are subjected.

3. *Club-foot, talipes equino-varus or total varus*

This is an effect of malposition, in which the lower limbs are in the cross-legged position. Its names are unsatisfactory, as the ancient term of club-foot only applies to the final result of that complete lack of treatment which must at one time have been common. In this the foot turns into a rounded

contracted lump, taking the weight of the body on its dorsum. I have seen it stated that the binding of Chinese girls' feet began because an ancient Empress had double talipes, and decreed that all her attendants should hobble about on the backs of their feet as she did. It seems to me as likely an explanation as any other of this highly irrational custom.

Equino-varus as a description suffers from the not inconsiderable disadvantage that in the severer degrees the feet are not in equinus but in calcaneus. I propose the term "total varus" to express the moulding inwards of the foot as a whole, including the heel and ankle, and so distinguishing it from metatarsal varus, in which only the fore-part is affected. It has the advantage of avoiding the upsetting effect on parents of the use of the ugly term "club-foot."

This deformity has five characteristics, apart from the obvious correspondence of the dorsal surface of the foot to the uterine curve.

(a) The muscles of the calf are always poorly developed, presumably from the pinning of the leg preventing proper movement and circulation. In unilateral cases the calf starts with about 1 cm. less circumference than the normal side, and owing to the natural tendency of the child to put the best foot foremost the disproportion tends to increase as growth proceeds.

(b) The peronei are always weak and inert, compared with the muscles on the inner side of the leg. I think this is due not only to their being stretched in the causative position, but also to uterine pressure on the peroneal nerve producing a kind of mild "crutch palsy." In any event, the resultant lack of muscle balance is one of the most intractable factors in the condition.

(c) In double cases one side is always about 20 per cent. more severely affected than the other. This is because in the cross-legged position it is obvious that the leg on the outer side gets more moulding force on it than the one it shelters. There is never exact equality in the deformity, and never a very severe one on one side combined with a slighter one on the other; the relation is always about that of 80 to 100.

(d) The deformity is never, in my experience, associated with a congenital dislocation of the hips and their abduction is always freer than normal, as in metatarsal varus. This rule, of course, only applies if the knees are flexed in the normal foetal position; it is quite common to find total varus talipes associated with C.D.H. when the knees have been hyper-extended during development.

(e) There is invariably compression dysplasia of the inner side of the ankle and foot. This accounts for the notorious tendency of the deformity to recur, even after complete reversal of the moulding into valgus, since the outer side of the foot and ankle always grows faster than the inner.

FIG. V. Right total varus talipes, showing the spasmodic unopposed action of the invertor muscles of the ankle.

4. *Mutual metatarsal moulding*

Pairs of feet are fairly often found in which the inner convexity of a metatarsal valgus fits snugly into the inner concavity of a metatarsal varus. Deformities of this kind are not severe and are not associated with abnormal abduction range of the hips. The assumption therefore is that both feet, though with their soles in the normal position against the uterine wall, have swivelled sideways together and moulded each other accordingly.

IRREGULAR TALIPES

A literally infinite variety of mouldings of the feet can be found associated with abnormal position of the limbs. All of them, however, have the characteristic convex moulding on the appropriate surfaces and very often show mutual moulding. Their association with arthrogryposis and spina bifida has already been noticed and explanations for this suggested.

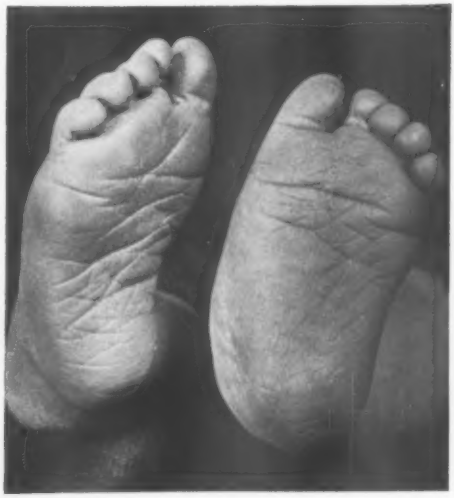

FIG. VI. Mutual deformity of feet. Note correspondence of convex inner border of left foot to concave inner border of right.

TREATMENT

This does not come into the scope of this paper, but some observations may be made.

1. As the original condition nearly always includes stiffening of the joints and atrophy of the muscles, and as these effects are notoriously produced by immobilization (e.g. in plaster after a fracture) it does not seem very logical to employ immobilization as a method of correction.

2. It is necessary to obtain true *over-correction*, e.g. turning of total varus into total valgus; changing one deformity into its opposite. A study of textbooks will show that the term over-correction is frequently used for simply bringing the foot into a normal walking position. In all the early years of its life the child with total varus should walk in extreme valgus, kept by wedges on the outer sides of the feet.

3. Bedclothes tend to reproduce the original deformity of total varus by turning the feet downwards and inwards. They must be countered by a splint that pulls the foot upwards and outwards.

4. Unbalanced muscle pull must be countered by exercises; if they are unsuccessful by tendon transplants.

5. The foot has a marked tendency to respond to cutting operations by throwing out masses of tough and contracting scar tissue. This means that what are known as "soft tissue releases" produce a reaction that in a few years leaves the area attacked more contracted than before.

6. Cutting tendons round the ankle is as disastrous as cutting them round the wrist. Once the tendo Achillis has been cut an even tolerable result is impossible.

OTHER HYPOTHESES OF CAUSATION

1. *Irregular action or insertion of muscles*

A very sufficient objection to this is that some of the most severe examples of talipes occur in infants in whom the muscles have never been active at all; spina bifida and arthrogryposis. It is hardly necessary after noting this to point out that many varieties of mouldings of the feet could not possibly be caused by any muscular pull.

2. Congenital dislocation of the head of the astragulus has been put forward as an explanation of club-foot. How this dislocation could mould the heel and ankle behind it is not discussed; and in any event this hypothesis gives no explanation of all the other varieties.

3. Duraiswami (1952) has asserted that he can produce talipes and dislocation of the hips in chickens by dosing the embryos with insulin. The obvious explanation of his results is that the insulin so wrecks the bony structures of the embryo that it cannot stand up to the normal forces of compression inside the egg. Much the same effects can be seen on the bones of new-born cases of osteogenesis imperfecta; and the talipes of arthrogryposis may also be compared.

4. *Persistence of early foetal positions*

This might serve as an explanation of club-foot alone, particularly if normal foetuses ever had their feet in a true club-foot position. But it is difficult to take it as seriously as is done in Germany when one considers the three regular forms and the infinite variety of irregular ones.

of parts of the body to be exposed to the deforming factors described; to develop in malposition or to be subjected to the two forms of pressure. But the argument of this paper is that a simple genetic failure of normal development, such as accounts for hare-lip or undescended testicle, is insufficient to explain the deformity as a whole, and that

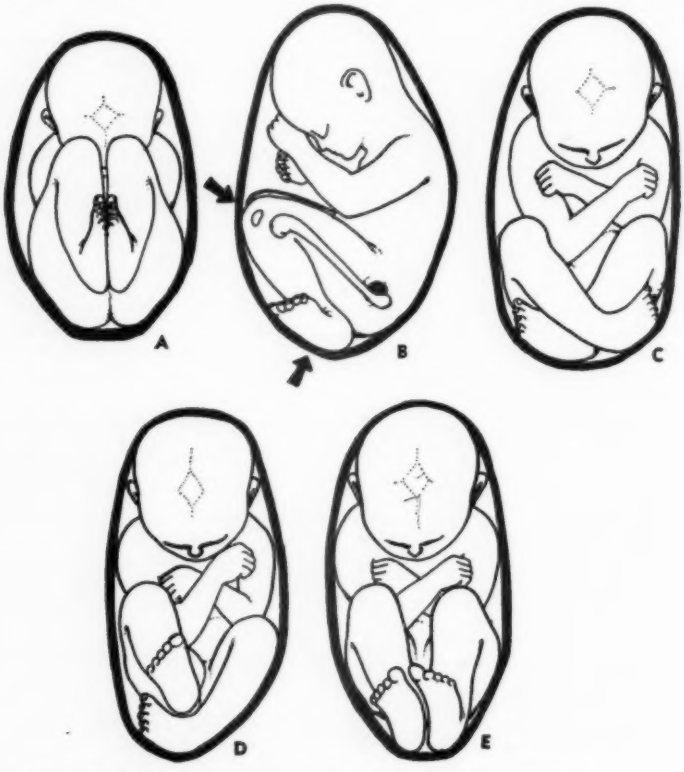

FIG. VII. Positions of foetus assumed to cause various forms of talipes:—
(a) Metatarsal varus, malposition.
(b) Metatarsal valgus, normal position with increased mechanical pressure. Arrows show direction of thrusts which are apt to dislocate the hip as well as forcing the foot round the outer side of the leg.
(c) Double "total varus" talipes. Note how foot on outer side must get more pressure than the one on the inner side.
(d) Single total varus talipes.
(e) Mutual metatarsal moulding talipes.

OBJECTIONS TO THE MOULDING HYPOTHESIS OF CAUSATION

1. *That there are racial, familial, and sexual variations in its incidence*

It is of course obvious that there must be a genetic factor which accounts for the tendency

to recognize the moulding factor in its production gives valuable understanding of its true nature. To illustrate it by an anology to the artificial deformities which have been mentioned the genetic factor corresponds to the chance of being born of a certain race, of a certain sex, at a certain time, and in a

certain social class; but all these factors merely expose the foot to certain outside moulding forces.

FIG. VIII. Arthrogryposis, showing how the stiffness of the elbows holds the hands against the uterine wall, and so produces a "talipes" in them. Note that undue size of uterus cavity (due to hydramnios) allows knees to stiffen in extension.

2. *That the moulding hypothesis does not account for every kind of congenital deformity*

It would hardly be necessary to mention this objection were it not so frequently made.

3. *The assertion that the deformity is not the result of the position, but that the position is the result of the deformity*

This is a purely verbal confusion, of a kind familiar to students of law and theology. The deformity and the position are the same thing, as can be seen by applying the same argument to the deformity of bandy legs; "the deformity of bandiness is not the result of the position of bandiness, but the position of bandiness is a result of the deformity of bandiness."

SUMMARY

A definition of talipes is suggested, and it is maintaned that if this definition is accepted and the different varieties of talipes analysed it can be demonstrated that the deformity is the result of moulding by mechanical forces in utero. Three regular varieties of talipes and an unlimited number of irregular ones are described.

FIG. IX. Premature infant, born dead, which had lumbar spina-bifida, Arnold-Chiara deformity, hydrocephalus, malposition of legs, dislocated hips and varus talipes.

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THE ROLE OF TESTICULAR VASCULAR ANATOMY IN THE SALVAGE OF HIGH UNDESCENDED TESTES

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AND

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THERE are a number of high undescended testes which cannot be brought without tension to the scrotum by conventional techniques of orchidopexy. Most of these testicles suffer an unsatisfactory surgical fate, whether by orchidectomy, abdominal reposition, multiple stage orchidopexy, or ill-advised efforts to secure scrotal fixation under tension. As a group, they present a special problem in management which has received only scant attention in the literature and to which no generally acceptable solution has been found.

The aim of the present investigation has been to refocus attention on high undescended testes and to find a means of reducing the heavy testicular wastage within this group. This paper presents our experience with one method of treatment by which their rate of salvage can be increased. Because the successful application of this method demands a more intimate knowledge of testicular vascular anatomy than is currently available in the surgical literature, this anatomy is presented in some detail. Its importance, moreover, is relevant not only to the mobilization of high undescended testes, but also to an unsuspected but avoidable source of wastage among those testes which can be brought without difficulty to the scrotum.

Blood Supply of Normal Testis

The two questions of foremost relevance to this present study are whether the testicular artery has anastomotic branches and, if so, whether these are functionally adequate for survival of the testis after ligation of the main testicular artery.

Anastomotic branches

It has long been known that the testicular artery is not anatomically an end artery, Jarich (1888), Colle (1902), and Picque and Worms (1909) have found that anastomoses are constantly present between the testicular (internal spermatic), vasal (deferential) and cremasteric arteries. More recently these observations have been confirmed by Harrison's arteriographic (Fig. 1), and microradiographic studies (Harrison and Barclay, 1948; Harrison, 1949).

Similar anastomoses have been described in the dog (Ellenberger and Baum, 1891; Griffiths, 1896) and can also be found in other mammalian testes (Harrison and Weiner, 1949). In animals, however, these anastomoses are never so well developed as they are in man (Harrison, 1949).

Testicular end-arteries

Harrison and Barclay (1948) have shown that no anastomotic vessels enter the substance of the testis directly from either the head or tail of the epididymis. Any anastomotic supply to the testis can reach it only by way of the main testicular artery or its major superficial branches proximate to the testis. They showed also that the septal branches of the testicular artery are end-arteries and that the manner in which these reach the parenchyma of the testis in no way resembles the classic descriptions. They are not, as has previously been taught, hilar arteries radiating from the mediastinum testis to the periphery. Instead, they arise from the major superficial branches which ramify over the body of the testis, most commonly as two vessels lying immediately beneath the tunica albuginea to form the tunica vasculosa. Sometimes there is only one major superficial vessel. When two are present they need not course separately over medial and lateral aspects of the testis but both may run together on one side or the other. In this superficial situation these vessels present an unexpected hazard to all or part of the blood supply of any testis which is anchored to the scrotum by direct tunical suture.

FIG. I. Plate 2, Figure 2, from Harrison (1949). "Radiograph of the left testis and spermatic cord from a man aged 71 after the injection of "Chlorbismol" into the vasal artery (v.a.). This artery enters into anastomosis with epididymal branches of the testicular artery (t.a.) and also directly with the testicular artery toward the inferior pole of the testis. The cremasteric artery (c.a.) is also injected. Natural size."
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From the tunica vasculosa the septal twigs of these superficial branches pass deeply along the septa and reach almost to the mediastinum. These septal end-arteries then double back upon themselves to take a recurrent and often spiralling course towards the gland periphery, supplying directly testicular tissue along the way. It is these terminal twigs and not the major superficial vessels which should be sacrificed in the course of either a biopsy or the special bleeding tests to be described.

Functional anastomoses

It is established therefore that the ultimate branches of the testicular artery are anatomical end-arteries, but as to whether the main trunk with its anastomoses behaves as a functional end-artery both the experimental and clinical evidence is conflicting.

(a) Experimental evidence

Two detailed reviews of the bulk of this evidence have already appeared (Koyano, 1923; Wangensteen, 1927) but present contradictory conclusions. No experimental work of note has appeared since then to help resolve the issue. Here it must suffice to say that for every bit of evidence that the testis atrophies after interruption of its main artery, there seems to be at least as much evidence that it can survive this interference.

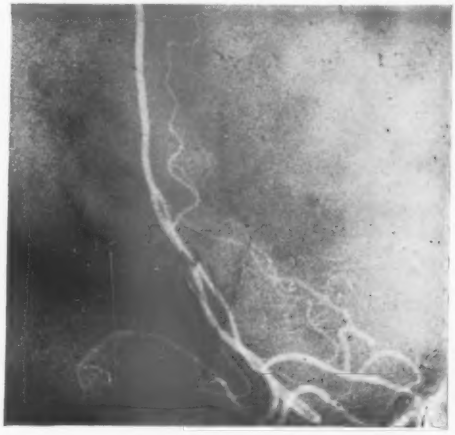
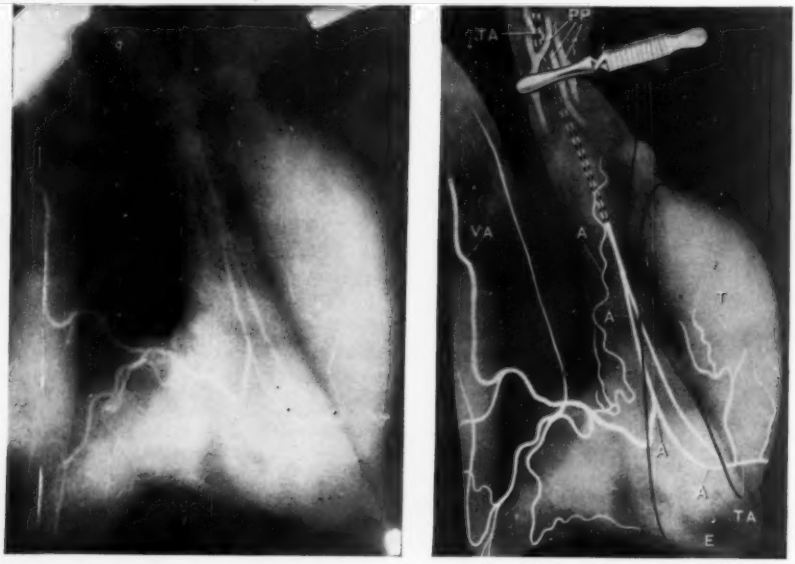


FIG. II. A.D., age 11 years and 2 months. Operative angiogram showing filling of vasal artery and branches to the epididymis and testis. The testis is visible as an oval opacity on the lower border of the radiograph.

A similar conflict can be found in the reported results of simultaneous ligation of the main artery and veins as compared with ligation of either alone. In the dog, for instance, both Koyano and Wangensteen agree that arterial ligation alone need result in no more than a minor degree of testicular atrophy, and spermatogenesis can continue. They present, however, completely contrary findings in regard to ligation of artery and veins together. Koyano found an even slighter degree of atrophy after this procedure than after arterial ligation alone, whereas Wangensteen reported that "ligation of the internal spermatic artery and pampiniform plexus always results in atrophy of the germinal epithelium and interstitium of the testis as well. Statements to the effect that ligation of these vessels is without deleterious effect on the testis will not bear investigation."

Wangensteen chose to accent the importance of this observation rather than the significance of his other findings that simultaneous partial venous ligation can result in a viable testis and that interruption of the veins in the pampiniform plexus interferes more with the circulation than high ligation of the testicular vein alone. This latter finding parallels some prior observations of Koyano (1923), the importance of which has been overshadowed in Anglo-Saxon literature by the weight of Wangensteen's somewhat sweeping pronouncement.

Koyano suggests that involvement of the cord vessels in wound scar can be a factor contributing to testicular atrophy. The closer to the testis that the incision is placed, the more likely is this cicatrization to take effect. The complications of haematoma or sepsis will of course increase such scarring. Sepsis, moreover, favours vascular thrombosis which, in turn, favours atrophy of the testicle. Koyano, therefore, discounts much of the early experimental work resulting in atrophy because non-sterile techniques or scrotal incisions were employed.



(a) (b)

FIG. III. M.D., age 11 years and 6 months. Operative angiogram — retrograde filling from the vasal artery.
(a) Photograph of angiogram, and (b) retouched photograph.
The testicular artery (T.A. lower) and sundry anastomoses (A) are filled from the vasal artery (V.A.), Testis (T.), Epididymis (E.).
The clamp is shown compressing the testicular artery (T.A. upper) and the pampiniform plexus of veins (P.P.); the dotted lines represent the unfilled segment of testicular artery above and below the clamp.

Koyano alone has deliberately sought an explanation of the wide variation in reported findings. In a series of carefully executed experiments in dogs and rabbits he showed that the nearer to the testis that the blood supply was interrupted, the greater was the resulting testicular atrophy. One rational explanation of this finding is that the nearer to the testis that the main vascular trunk is ligated the fewer are the anastomotic arcades available to supply the ultimate testicular end-vessels. As an additional factor,

Likewise in regard to atrophy we must say that because of the greater richness of anastomoses to the human testis (Harrison, 1949), none of the results of animal experiments is necessarily applicable to man. Such experiments serve to emphasize how great is the price of indiscriminately sacrificing testicular vessels, but every contrary finding of testicular survival urges us to seek all the possible factors favouring survival of the testis in man. Of all the reported experimental studies those of Koyano appear to have been the

most critically conceived and executed. His findings and conclusions offer us not only a strong pointer as to what these factors may be but also some hope for their practical clinical application. They strongly suggest that the secret of testicular survival lies in the high ligation of testicular vessels — ligation at a level where the anastomotic branches are least likely to be injured, by either the manipulations or local complications of surgery. It remains to be seen what clinical evidence can be found in support of this approach and of the functional adequacy of the anastomotic supply in man.

(b) Clinical evidence

Evidence for the adequacy of the anastomotic supply in man comes mainly from two sources — operations for the radical cure of varicocele and of recurrent inguinal hernia. Koyano (1923) and Wangensteen (1927) again have reviewed the many early experiences with these procedures, whereas the few scattered reports to have appeared since then have been reviewed more recently by Maluf (1957). The results have varied from total atrophy to survival of apparently normal testes.

Typical of such experiences are those of Burdick and Higginbotham (1935) who observed the subsequent fate of 79 testes whose main vessels had been severed in the inguinal canal for the radical cure of inguinal hernia. Of these 79 testes 42 remained normal in size, in 11 there was slight atrophy, 11 were half or less of normal size, and 15 had completely atrophied. Since testicular salvage was not the primary intent of this undiscriminating vessel division, it is interesting to compare these findings with those of Palomo (1949) who deliberately practised high extraperitoneal section of the vessels above and lateral to the inguinal canal. He found no subsequent atrophy in 40 testes submitted to this procedure for the cure of varicocele.

Maluf (1957) presents further evidence in man that discriminate high division of the vessels permits testicular survival. This he observed after high testicular artery division during such diverse procedures as nephrectomy for renal carcinoma, orchidopexy, pyeloplasty and block dissection of juxta-aortic nodes. He found the testis atrophied, however, after section of both testicular and vasal arteries during radical cystectomy. It should be noted in passing that in all the foregoing clinical reports the fate of the testis was estimated on clinical grounds alone. Maluf, on the other hand, is the first to produce biopsy evidence of the survival of both normal and undescended testes after division of the testicular vessels in man.

We can now say with certainty that the main testicular artery in man is not of necessity, as we have been led to believe, a functional end-artery. The clinical evidence seems strongly to support what Harrison's anatomical studies suggest — that the normal adult human testis has what should be in most cases an adequate anastomotic supply. The clinical evidence is likewise in keeping with the lessons learned from animal experiment — that the fate of the testis depends not only on this anastomotic supply but on whether this supply is interfered with by the technique and level of main vessel division.

BLOOD SUPPLY OF UNDESCENDED TESTES

Although there is now sufficient evidence of functional anastomoses to normal testes, no satisfactory evidence is yet forthcoming that the same is true for undescended testes. Harrison, in fact, was careful to point out that his findings were not necessarily applicable to undecended testes, and, furthermore, that the post-mortem demonstration of anastomotic vessels was more difficult in the testes of children than of adults. Further investigation was therefore nesessary into the vascular anatomy of undescended testes and the results of their main vessel division. Indirect evidence of an anastomotic blood supply to undescended testes is already available in the literature but can now be supplemented by the more direct evidence obtained in this present study.

(a) *Previous evidence*

Again, in regard to undescended testes, the reported results of main vessel division have varied from complete atrophy to survival of testes of satisfactory size. Mignon (1902) appears to have been the first to practise division of testicular vessels for the correction of cryptorchidism, and presented three cases without atrophy. Bevan (1903) also

claimed satisfactory results from this procedure which he performed on approximately ten per cent. of the undescended testes for which he operated. Moschcowitz (1910) performed this operation on 22 testes with subsequent atrophy in only one. However, Mixter (1924) reported atrophy of 13 out of 15 testes in which all the vessels were cut and 5 out of 7 testes in which only some of the vessels were cut. Wangensteen (1927) condemned the practice on the basis of his experimental findings in dogs, and McCollum (1935) reported that every patient operated on by this technique at the Children's Hospital, Boston, showed total testicular atrophy on follow-up ten to twenty years later.

In the absence of any precise knowledge of the vascular anatomy of undescended testes, it seems unlikely that this procedure would have been employed with any greater discrimination by the above authors than by those who were using it for hernia or varicocele. It is not so surprising, therefore, in view of the unpredictable fate of the testis, that this operation fell into disrepute. There must be an explanation, however, of why this method succeeded in numerous cases, and we are therefore led to seek in the vascular anatomy of such testes a rational basis for the more discriminating application of the method.

(b) *Evidence from this study*

As far as we can discover, no anatomical studies have yet been reported on the blood supply of undescended testes. Even in normal scrotal testes, the post-mortem demonstration of vascular pattern is difficult in childhood. On this account we have thought that a more fruitful approach would be a deliberate attempt to obtain at operation direct evidence for the presence or absence of arterial anastomoses to undescended testes.

Visible anastomoses

That there frequently are such anastomoses was suggested at operation by direct inspection of the transilluminated cord structures, in which there often appeared to be an anastomotic arcade of vessels running marginal to the epididymis between the testicular and vasal arteries.

More direct evidence of an anastomotic supply in a majority of high undescended testes was found during operation on 12 testes in 11 patients. This evidence was obtained either by testicular angiography, by bleeding tests or both.

FIG. IV. M.D., age 11 years and 6 months. Operative testicular angiogram from the same patient as in Fig. III, showing orthograde filling of the main testicular trunk and various fine anastomotic vessels, including what is probably the vasal artery. The testis is the pale oval opacity occupying the upper half of the radiograph.

Testicular angiography

The only report published so far of a method of operative testicular angiography (Brodny *et alii*, 1955) leaves much to be desired. This method describes direct needle injection into the isolated testicular artery, found by dissection among spermatic cord structures through a small scrotal incision. Anyone who has attempted, during orchidopexy, the isolation of this artery as a separate structure knows that it is easier to feel than to find. Difficulties of identification and technique must all militate against the use of this method, which has not yet yielded radiographs which unquestionably show the arteries with clarity or in detail.

By trial and error a technique of operative testicular angiography was evolved which gave us direct evidence of an anastomotic blood supply in several undescended testes.

Details of operative testicular angiography

During the course of orchidopexy the transversalis fascia was routinely opened and split widely from the internal to the external ring. The inferior epigastric artery, thereby exposed, was then divided with two-fold purpose: firstly, this section subsequently ensures the greatest possible length and mobility to the vas and its vessels; and, secondly, the stem of this artery affords a route of entry for the arterial catheter.

A polythene catheter (No. 2 Allen and Hanbury) was passed through the stem of the inferior epigastric artery to the external iliac artery and upwards from thence to any predetermined level in aorta or iliac vessels. The desired level was readily achieved with the help of preliminary radiographic films of the dye-filled catheter.

The catheter tip, on one occasion, was arranged to lie just proximal to the origin of the testicular vessels (Fig. X, level No. 1). The injection of dye from this position achieved orthograde filling of the testicular artery together with several auxiliary channels (Fig. IV). Evidence, however, of flow through these channels, with the main testicular artery occluded, was the chief concern of this project, so that our efforts were subsequently turned to a retrograde method of filling. The artery to the vas takes its origin from one or the other vesical branch of the internal iliac artery (Cunningham, 1951). For the successful display of these vessels the catheter tip was set to lie just distal to the aortic bifurcation and proximal to the internal iliac origin (Fig. X, level No. 2). Immediately prior to the injection of dye a small bulldog clamp was placed temporarily across the testicular vessels, leaving the vas and its vessels free.

Films were exposed from two to four seconds after the injection of 60 per cent. "Urokon" solution in amounts varying from 10 to 20 cubic centimeters. With a lead shield guarding the opposite testis, simultaneous exposures of two films were made — a small dental film directly beneath the testis, and a large plate beneath the patient to reveal the extent of filling of the major vessels. In some large films there was excellent internal iliac filling but no vessels appeared on the testicular film. In others, inadequate internal iliac filling may have been due to escape of dye into the leg. In later trials, to limit this escape, a sphygmomanometer cuff on the thigh was inflated to pressures above the arterial, but without notably increasing the frequency of vasal artery filling. Among all these variables it was not possible to discern, from the small series attempted, which if any was the limiting factor in the success or failure of the venture.

At first, it was hoped that this angiographic technique would provide the key to those testes which could survive division of their main testicular vessels. However, out of seven of these patients in whom testicular angiography was attempted, in only four patients was the procedure successful enough to achieve demonstrable filling of any spermatic cord vessels. In one of these only the vasal artery was filled. In one, the artery to the vas was filled, together with branches from it to the epididymis and testicle (Fig. II), and in two cases retrograde filling through the vasal artery of the main testicular artery was achieved (Fig. III). This display in one patient was further supplemented by the demonstration of anastomotic and vasal vessels by orthograde filling from the main testicular trunk (Fig. IV).

Curiously enough, our first two attempts at angiography were the most successful, otherwise we should not have persisted with our efforts in a procedure which is technically irksome and adds considerably to the time of operation. The cause of our subsequent failures is by no means clear but it is obviously not a technique which can be adapted readily to routine use. In its place we have evolved the "bleeding tests" — a simpler method of safely demonstrating, prior to division of the testicular vessels, an anastomotic supply to any particular testis.

Bleeding tests

By blunt dissection of the thin transparent veil of tissue between the testicular and vasal pedicles a window is made in the spermatic cord. A small bull-dog clamp is next placed across the testicular vessels (Fig. IX). A short biopsy incision is then made

TABLE 1

RESULTS OF ORCHIDOPEXY BY MAIN TESTICULAR VESSEL DIVISION IN THE TREATMENT OF HIGH UNDESCENDED TESTES

Analysis of Details of Twelve Cases

Patient	*Type of vas*	*Side*	*Age at opn.*	*Location*		*Size*		*Months follow-up*	*Biopsy months post-op.*	*Remarks*
				At opn.	*Post-op.*	*Pre-op.*	*Post-op.*			
J.D.	"Long loop type"	L	13	Abdominal	M.S.	Normal	Slightly Small	12	—	Successful salvage
D.M.	"Long loop type"	R	13	Abdominal	L.S.	Small	Very small	22	22	Unsatisfactory result Biopsy: Partial survival, partial atrophy
P.Y.	"Long loop type"	L	1	At internal ring	H.S.	Very small	Very small	5	—	Abnormally shaped hypoplastic testis at operation. Unsatisfactory result
K.B.	"Long loop type"	L	11	Canalicular	L.S.	Slightly small	Slightly small	18	18	Successful salvage. Biopsy: viable testis.
M.D.	"Long loop type"	R	11	Canalicular	M.S.	Normal	Slightly small	17	17	Successful salvage. Biopsy: viable testis: successful orthograde and retrograde angiography
D.H.	"Long loop type"	R	7	Canalicular	L.S.	Normal	Normal	21	6	Successful salvage. Biopsy: viable testis: successful retrograde angiography
	"Long loop type"	L	9	Canalicular (Incarcerated)	M.S.	Normal	Normal	14	—	Successful salvage
I.C.	"Adequate in Length"	L	14	At internal ring	L.S.	Normal	Normal	7	7	Successful salvage. Biopsy: Differentiation to spermatocyte stage in many tubules: Leydig cells present
M.G.	"Adequate in Length"	L	8	Canalicular	M.S.	Normal	Normal	5	—	Successful salvage. Vasal A filling on angiography
A.D.	"Adequate in Length"	L	11	Ectopic in S.I.P.	L.S.	Slightly small	Slightly small	4	—	Successful salvage. Successful retrograde angiography
P.O.	Very short	R	7	Canalicular	—	"Small and Atrophic	Total Atrophy	7	—	Unsuccessful operation. Bleeding test not performed
A.B.	Not noted	R	9	Emergent at External ring	—	Small "Flabby"	Total Atrophy	8	—	Unsuccessful operation

Abbreviations
H.S. = High scrotal position
M.S. = Mid scrotal position
L.S. = Low scrotal position
S.I.P. = Superficial inguinal pouch

through the tunica albuginea, and if an anastomotic supply exists, bright arterial bleeding will be observed for several minutes from the cut-terminal twigs of the testicular artery. This sign of an anastomotic supply can be confirmed subsequent to division of the main testicular vessels by noting whether the bright arterial flow from the distal cut end of the testicular artery will likewise continue for several minutes. This bleeding test was performed on 11 out of 12 of the testes reported on in this paper and was positive in each case tested.

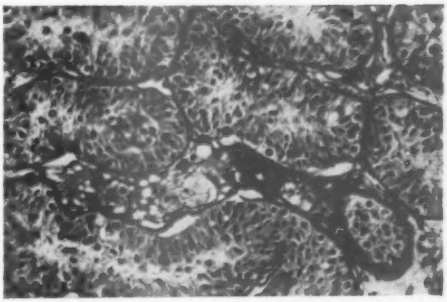
In spite of our technical failure to produce arteriographic confirmation in every case, from the direct evidence of these bleeding tests we conclude that there is an anastomotic arterial supply to a majority of high undescended testes. This contention is further supported by the evidence, now to be presented, of survival of a majority of high undescended testes subjected to division of the testicular vessels.

CLINICAL MATERIAL AND RESULTS (TABLE I)

The 12 undescended testes which form the basis of this report were testes which could not be brought without tension to the scrotum until after division of the testicular vessels.

Clinically all but the two furthest descended testes belonged to the group of true cryptorchids (Mimpriss, 1952) in that during repeated examinations they could not with certainty be felt outside the inguinal canal.

Surgically, according to their position at operation, these twelve may be classified as follows: abdominal, 2; canalicular, 8: emergent, 1; and superficial inguinal, 1. Some doubts may be felt about the propriety of including all these testes, especially the latter two, in the category of "high undescended testes", since undoubtedly many more highly placed testes have at times been brought with success to the scrotum. For convenience, however, the whole 12 have been dubbed with this title on the basis of the shortness of their cord structures. These testes resisted scrotal placement even after thorough dissection extending as far as possible from the internal ring along the retroperitoneal course of the vessels. The testis with the furthest degree of descent actually provided one of the worst clinical results of the series.



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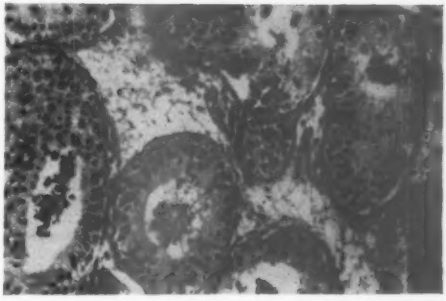

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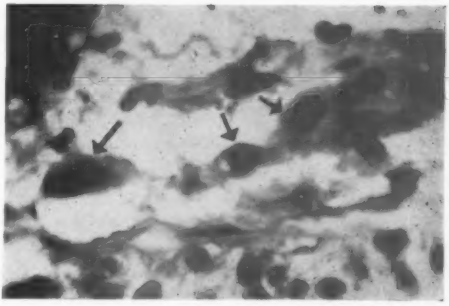


(c)

FIG. V. M.D. Photomicrographs of biopsy specimens of right testis.

(a) At time of orchidopexy when the testicular vessels were divided at age of eleven years and six months. (x 250)

(b) Seventeen months later: testis in scrotum: note survival of tubules. (x 250)

(c) Showing healthy Leydig cells (arrows) in another area of the same specimen as (b). (x 1120)

Clinical results (Table I)

It has been possible to follow up these 12 testes for periods varying from four to twenty-two months.

Eight out of 12 testes were regarded as good anastomical results on the basis of their satisfactory size, shape, consistence and degree of dependency in the scrotum.

Four testes were classified on clinical grounds as unsatisfactory results: two of these had completely atrophied; the third, a hypoplastic and abnormally shaped testis at operation, remained subsequently as a small nodule of unchanged size palpable high in the scrotum; the fourth testis was high in the scrotum and smaller in size than its fellow, although biopsy twenty-two months after operation showed surviving testicular tissue with a few areas of apparent tubular atrophy.

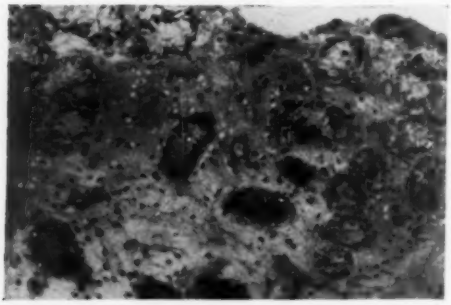


FIG. VI. D.H., age 8 years. Photomicrograph of biopsy specimen of scrotal testis six months after orchidopexy using the technique of division of the testicular vessels — note survival of tubules. (x 250)

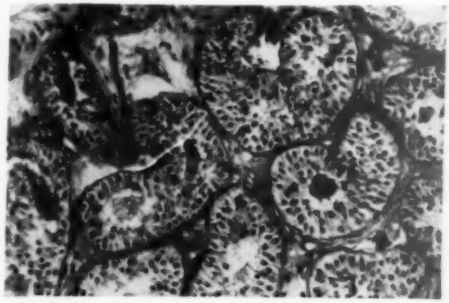
In those testes in which any appreciable degree of atrophy occurred, the result was apparent by the end of the third week. No case was regarded as successful until it had been observed well beyond this early post-operative period.

Biopsies

In the two cases of complete clinical atrophy no attempt was made to obtain biopsies.

Biopsies were taken from 4 testes regarded clinically as good results. These four biopsies showed viable testicular tissue at periods of six, seven, seventeen and eighteen months respectively, after division of the testicular vessels (Figs. V, VI, VII and VIII).

The histological appearances are not those of normal testicular tissue: the tubules are smaller, the lining epithelium is relatively undifferentiated and the interstitial tissue is increased in amount. Comparison of the



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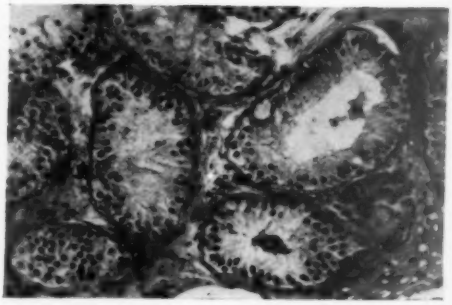


(b)

FIG. VII. I.C., age 14 years. Photomicrographs of biopsy specimens of right and left testes obtained at the same time.

(a) Right undescended testis showing tubules typical of this condition. (x 250)

(b) Left scrotal testis, seven months after orchidopexy using technique of division of testicular vessels. (x 250)

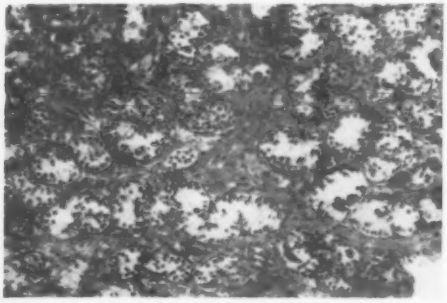
Note survival of tubules.

operative and post-operative appearances shows an increase in differentiation commensurate with the development to be expected during the time interval between biopsies (Figs. V, VIII). These appearances are, however, quite compatible with the histological features (Fig. VIIa) of cryptorchid testes of equivalent age (Robinson and Engle, 1954; Charny and Wolgin, 1957).

This histological proof that certain cryptorchid testes can survive division of the main

testicular vessels confirms a similar observation, the first of its kind, in one case only, by Maluf (1957). Of particular interest to note is the fact that the Leydig cells can also survive this vascular interruption (Fig. Vc).

In attempting to predict which testes will and which will not survive this interruption, there were no guides to help us at the outset of this investigation. With the further evolution of the study, however, it has become increasingly clear that our share of poor results could have been reduced by a fuller awareness of all the relevant features of the surgical anatomy of these testes.



(a)

(b)

FIG. VIII. K.B.: Cryptorchid. Photomicrographs of biopsy specimens of left testis.

(a) Initial biopsy specimen at time of orchidopexy at age of eleven years. (x 250)

(b) Biopsy specimen of scrotal testis eighteen months later showing survival and further development of the tubules. (x 300)

APPLIED SURGICAL ANATOMY

Of foremost importance in conserving the maximum number of anastomotic vessels is the level of division of the main testicular trunk. Because anastomotic vessels may join with the main trunk some little distance above the testis, the testicular vessels should not be divided too close to the testis but always as far from it as possible. This principle is based primarily upon the evidence, already reviewed, of experiments in animals and operations for varicocele in man. Nevertheless, reference to Figs. III and IX shows that it should also be adhered to for undescended testes. The angiogram shows clearly that interruption of the testicular vessels much beyond where the clamp was placed would have involved the sacrifice of several fine anastomotic channels.

Of at least equal importance to the above precept are certain features of the vas and its associated vessels which affect the selection of cases suitable for the method and some of the finer technical details in its execution.

With undescended testicles one sometimes finds that although the testicular vessels themselves are short, the vas deferens and its companion vessels are long, and run a recurrent course (Fig. IX). The long vas and its accompanying vessels emerge through the external ring and may extend further either to the superficial inguinal pouch or scrotum before looping back to rejoin the testis. We have called this the "long loop" type of vas. This particular variant of the surgical anatomy has previously been recorded (Moschcowitz, 1910; Mixter, 1924) but without its practical significance being noted.

It is this "long loop vas" type of undescended testis (Fig. IX) which, we believe, lends itself most admirably to orchidopexy involving testicular vessel division. By careful dissection, aided by transillumination, this long loop of vas and its companion vessels can be unravelled. The extra cord length thereby gained permits the testis to be placed in the scrotum.

In this unravelling process numerous strands of tissue or "mesentery", which may include one or more fine anastomotic vessels, may have to be divided. Here again the bleeding test is of fundamental importance in deciding which of these strands may be safely cut. If each such strand is gently clamped and bleeding still continues unabated

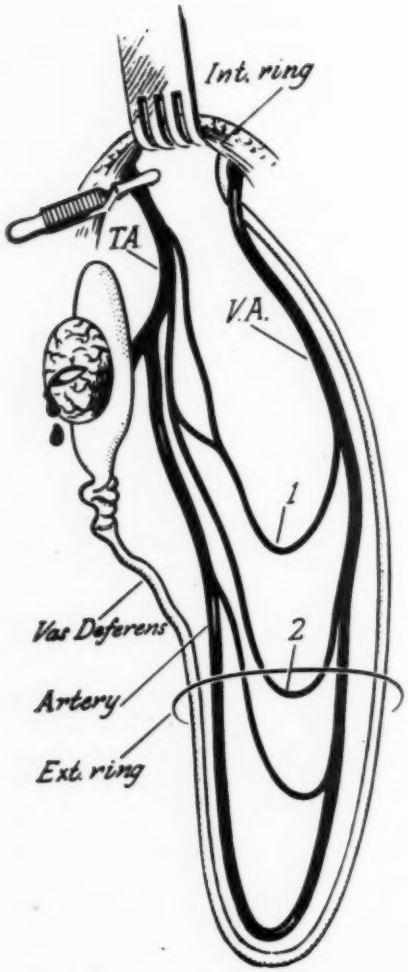


FIG IX. Diagram to show—

(a) The "long loop" vas with the undescended testis lying at the internal ring. The artery to the vas (V.A.) runs a long recurrent course with several anastomotic branches (1 and 2) (the accompanying veins are omitted from the diagram).

(b) Technique of bleeding tests — the testicular artery, with veins (T.A.), is compressed with bulldog clamp cranial to testis: the tunica vasculosa is incised: continued bleeding from this incision indicates adequate auxiliary blood supply to testis; high ligation of testicular vessels is safe. Leave cut caudal end of T.A. to bleed for further tests: place bulldog clamp at point 1; if bleeding continues it is safe to divide this branch. This affords further mobilization of testis. Repeat procedure at point 2 if necessary. Ligate T.A., and suture incision of tunica.

from the distal cut end of the testicular artery, the strand may be safely cut. Not even such minor twigs, however, should be sacrificed unless their division is essential to obtain adequate cord length.

In the 12 testes reported here (Table I) the following anatomical features were found: the long loop type of vas was noted in 7 cases; in 3 instances the vas was described as being "adequate" in length; in one case the vas and its associated vessels were extremely short; in one other case the length of the vas was not specifically noted. These latter two testes atrophied completely.

Two types of cryptorchid testes are anatomically unsuited to this method of orchidopexy — first, the testis with a very short vas deferens as well as short testicular vessels, and secondly the grossly hypoplastic type in which there can be no certainty that vascular anastomoses are present. The failures in our group we judge in retrospect to belong to these categories and for these reasons were unsuited to the operation described.

DISCUSSION

The importance of the intimate vascular arrangements of the testis is emphasized here for two reasons: this anatomy not only provides a key to the conservative surgery of high undescended testes but also has a bearing on the overall problem of testicular wastage following orchidopexy in general.

Some of this general wastage may be avoided by an awareness of the superficial distribution in the tunica vasculosa of the major subdivisions of the testicular artery. Careful preliminary inspection of these surface vessels will minimize the risk of their injury during the placement of anchoring tunical sutures or biopsy incisions. Many surgeons have felt for some time that the highest incidence of testicular atrophy is encountered following operations of the Torek type and Hand (1956) presents objective evidence in support of this opinion. Certainly the vulnerability of the vessels in the tunica vasculosa makes it advisable to abandon all methods of scrotal anchorage involving multiple tunical sutures.

Considering now the major concern of the presentation, it is necessary to evaluate the

role of applied vascular anatomy in the conservation of high undescended testes. The salvage of eight out of twelve such testes in our group represents a modest though promising measure of achievement. Comparison with other published figures is difficult because of the very varied claims which have been made for the overall success of orchidopexy in the treatment of undescended testes generally. These claims range as widely as from 16 to 100 per cent. success with an average close to 60 per cent. (Bishop, 1945).

These figures seldom include the cases in which orchidectomy was employed because of the impracticability of performing an orchidopexy (Mimpriss, 1937). In one of the few reports which does so (Hand, 1956), orchidectomy had sometimes to be resorted to even for emergent or ectopic testes.

It is hard to find specific accounts of the degree and incidence of post-operative testicular atrophy. As Denis Browne (1949) says: ". . . the frequency of testicular atrophy after operation needs far franker recognition than it has yet had." Mixter (1924), for instance, found total atrophy resulting from 9 out of 68 of his orchidopexies in which the vessels had not been cut. Bjerre (1936) after 251 orchidopexies, found good results in 58 per cent., bad results in 32 per cent., and total atrophy in 10 per cent. Aird and Wallace (Aird 1957) found that the testis was impalpable in more than half of patients in whom orchidopexy had been performed five years or more previously for unilateral incomplete descent. More fortunate experiences are the 2 per cent. incidence of atrophy encountered by Eisenstaedt (1950) and the 2.3 per cent. reported by Snyder and Chaffin (1956). Gross and Jewett (1956) claim that "about 90 per cent. of all patients operated on can be given a good result", and that atrophy "is a complication that can be reduced to a very small figure (5 per cent. or less) by the use of a correct procedure and a very careful technique in performing this." Details concerning the poor results are conspicuously lacking in all these accounts, especially in relating the fate of the testis to its observed level of descent. It is impossible, therefore, to evaluate the significance for undescended testes in general of either the most or the least optimistic of these figures. Even less can they help us to gauge the prognosis for the minority of surgically difficult testes specially selected here.

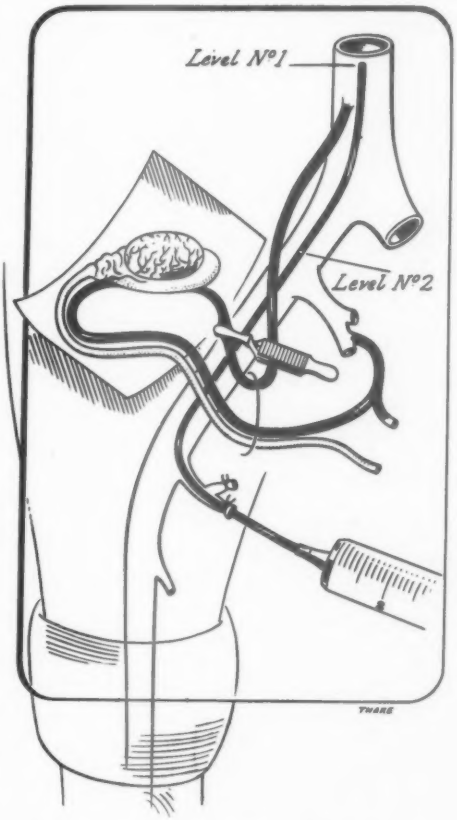


FIG. X. Diagram to show the technique of operative angiographpy of the testis.

The undescended testis was exposed and the inferior epigastric artery was isolated and cannulated with polythene tubing which was guided into the common iliac artery and aorta. Urokon 60 per cent. was injected at two levels. Orthograde filling of testicular artery occurred after injection at level No. 1 (no clamp on testicular vessels). Retrograde filling of testicular artery occurred through the artery to vas at level No. 2 (clamp placed across testicular vessels).

A dental film was used to show the angiogram of the testis and a larger film showed the great vessels simultaneously. Radiographs were obtained after injection at both levels or, more often at level No. 2 only (Fgs. II, III and IV).

Only a few accounts, such as those of Mimpriss (1952), Hand (1956), and Brunet, de Mowbray and Bishop (1958) deal with

the fate of the testis in relation to the particular level of arrest. From these it appears that the prospects are dismal for testes which have been impalpable clinically, successful scrotal anchorage being achieved in less than half of this group. Mimpriss, in fact, reported frankly that he treated by excision the probable failures in this group and that good anatomical results were achieved in less than half of the remainder. It is against this general and particular background of testicular wastage that our results in this present series should be considered.

In this series of 12 undescended testes which fell into the surgically difficult category, 8 testes have been salvaged. We feel that these results are sufficiently encouraging to justify our resurrection of this discredited method of orchidopexy by testicular vessel division. It is hoped that they will be sufficiently encouraging for others to apply the method, as an alternative to orchidectomy or abdominal reposition, in anatomically appropriate cases. It is likely that the method will again be discredited if due regard is not given, as occurred in some cases in this series, to all the relevant features of the applied surgical anatomy.

We report here only the early survival from surgery of a number of testes subjected to main vessel division with good anatomical results. No evidence will be forthcoming for some time as to the functional capacity of these testes or their ability to withstand prolonged gonadotrophic stimulation.

In bilateral undescent, this form of orchidopexy with division of the main testicular vessels is indicated in properly selected cases if we believe that the attainment of the scrotal position is likely to improve the fertility of the testis.

In unilateral cases, on the other hand, improvement of fertility cannot be claimed as an indication for operation. Of the several remaining indications for operation however, such as discomfort, attacks of pain, risk of torsion, relief of hernia, cosmetic and psychological reasons, satisfactory orchidopexy will achieve the desired relief of symptoms.

Admittedly, no easy solution presents to the problem of the high undescended testicle. We suggest that it is in the management of unilateral high undescended testes that orchidopexy by main vessel division will find its greatest application. Certainly its application will lead to the salvage of testes of the "long loop vas" type which could otherwise be lost and the method appears, in unilateral cases, as a more attractive choice than orchidectomy, abdominal reposition, or multiple stage procedures.

SUMMARY AND CONCLUSIONS

This paper presents a critical re-assessment of the treatment of high undescended testes by a method of orchidopexy involving division of the main testicular vessels. This appraisal is based upon the vascular anatomy of the testis which displays two main features of practical surgical significance: one is the pattern of arterial anastomoses to the testis and the other is the manner of distribution to testicular tissue of the subdivision of the main testicular artery.

The major subdivisions of this artery enter the substance of the testis, not at the mediastinum as is generally supposed, but peripherally from their superficial course over the body of the testis. Here, during orchidopexy, they present an unrecognized hazard to all or part of testicular blood supply. Careful inspection of the course of these vessels should be a routine preliminary to the placement of biopsy incisions or tunical sutures. Those methods of scrotal fixation of the testis involving multiple tunical sutures must increase the risk of post-operative testicular atrophy.

In regard to the pattern of arterial anastomoses there are constant collateral communications from the vasal and cremasteric arteries, of which the vasal contribution is the most substantial. None of these collaterals enters the substance of the testis directly but only by way of the main testicular artery. Dividing the main artery close to the testis, therefore, will at the same time cut off this anastomotic supply. High division of the testicular vessels on the other hand will preserve the collateral channels and, provided that these are functionally adequate for the purpose, permit survival of the testis.

The functional adequacy of these anastomoses have previously been explored in animal experiments and by operations for

varicocele, inguinal hernia and undescended testicles in man. The findings have been contradictory, however, in both the clinical and experimental fields. One reason for this confusion is that the importance of high division of the main testicular vessels has not been generally recognized. An additional factor is that the anastomotic vessels in animals are less well developed than they are in man. In review, the bulk of this clinical and experimental evidence favours the belief that when the testicular vessels are sectioned in such a way that the collateral channels are conserved, the anastomotic supply is functionally adequate to maintain viability in a majority of testes.

New evidence has emerged from this current investigation showing that functionally adequate anastomotic vessels are also present in a majority of high undescended testes. Again, the most important contribution is from the artery to the vas. The presence of such an anastomotic supply can be simply and safely demonstrated at operation, prior to division of the testicular vessels, by the "bleeding tests" introduced and described in this paper.

By means of these bleeding tests an anastomotic circulation was demonstrated at operation in 11 out of 12 high undescended testes and confirmed in 3 of these by a new technique of operative testicular angiography.

The functional adequacy of these vessels to maintain survival of the testis was apparent clinically from the good anatomical results observed in 8 out of 12 testes submitted to orchidopexy by main vessel division. This clinical evidence was confirmed in four cases by the demonstration of viable testicular tissue in subsequent biopsy specimens. Biopsy sections from one other testis, which clinically was small and high in the scrotum, revealed areas of surviving testicular tubules.

This degree of success, in testes which could not otherwise have been brought to the scrotum, is held to justify our reintroduction of the previously discredited method of orchidopexy by division of the main testicular vessels. The method is not to be used indiscriminately but only for the anatomically appropriate cases in the surgically difficult group defined.

For the selection of cases suitable for this procedure the demonstration of vascular anastomoses to the testis is not in itself sufficient. These vessels must also have an appropriate length for successful orchidopexy. The anatomically appropriate cases are those with the "long loop type" of vas and accompanying vessels. By scrupulous adherence to the surgical technique we have described, this long loop of vas and vessels can be unravelled to provide a vascular pedicle of sufficient length to place the testis in the scrotum. Integral aspects of this technique are transillumination of the cord structures, further utilization of the bleeding tests to show to what extent this dissection can safely be carried, and ligation of the main testicular vessels as far proximal to the testis as possible.

It is probable, both in the past and in this present series, that failures attending this type of orchidopexy were due to an incomplete appreciation of all the relevant features of the applied surgical anatomy. Grossly hypoplastic testes and those with shortness of all their cord structures we consider unsuitable for orchidopexy by this method.

The final physiological fate and spermatogenic potential of the testes operated upon in this series is as yet unknown. On the basis, however, of all the evidence presented here, orchidopexy by main vessel division is advocated as an attractive alternative to orchidectomy, abdominal reposition, or multiple stage orchidopexy in anatomically appropriate cases of high non-descent.

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RHEUMATOID ARTHRITIS, LEG ULCERATION AND POSITIVE LUPUS ERYTHEMATOSUS CELL TEST

REPORT OF A CASE OF SUCCESSFUL GRAFTING DURING CORTISONE THERAPY

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AND

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IN 1957 Allison and Bettley reported six cases of rheumatoid arthritis, chronic leg ulceration and a positive L.E. cell test. They noted the similarity in site and appearance of the ulcers, and suggested that these cases may represent variants of the systemic lupus erythematosus syndrome. The present case clinically resembles those previously described —a split skin graft applied to the ulcer was rejected, whereas a second graft applied during cortisone treatment was accepted, and survived under continuing cortisone administration.

CASE REPORT

A male pensioner, aged 75 years, presented with severe rheumatoid arthritis and extensive ulceration of the left leg. He had suffered no other illness, there being no allergic or vascular diseases. Rheumatoid arthritis had begun in 1943 and had slowly progressed resulting in severe deformity of the joints with crippling. Reddening and itching of the anterior aspect of the left leg had been present for six weeks followed by ulceration. There had been no antecedent trauma, claudication, sensory impairment or dependent oedema.

Examination revealed chronic rheumatoid deformities and painful limitation of movement of the wrists, knees, ankles and the small joints of the hands and feet. Large subcutaneous rheumatoid nodules were present over both elbows. On the medial aspect of the lower third of the left leg there was a large irregular ulcer, 15 x 10 cm. in area, with clearly defined edges (Fig. I). The base was granulating and clean except for some flakes of adherent fibrin. The posterior tibial and dorsalis pedis pulses were palpable. There was no evidence of varicose veins, or neurological disease. Blood pressure was systolic, 140; diastolic, 90 mm. of mercury.

Laboratory investigations: Haemoglobin 13 g. per 100 ml.; leucocytes 4,500 per c.mm.; erythrocyte sedimentation rate 35 mm. in 1 hour (Westergren); serum albumen 3.7 g. per 100 ml., globulin 2.9 g., gamma globulin 0.8 g.; cephalin flocculation test negative; Wassermann reaction negative. The autoimmune complement fixation (AICF) test (Gajdusek, 1958) was repeatedly positive to a titre of 1 : 8 to 1 : 16. A positive L.E. cell test was obtained on two occasions using the clot method of Magath and Winkle (1952).

Progress. On 9 Aug., split skin grafts (2 x 2 cm.) from the left thigh were applied to the clean base

FIG. I. Case of rheumatoid arthritis, chronic ulceration of the leg and positive L.E. cell test. The ulcer on medial aspect of the leg before grafting was undertaken.

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of the ulcer. No cortisone was given on this occasion. All grafts initially survived and epithelialization was complete in two weeks. The grafted area was carefully supervised, the left leg being dependent for increasing periods each week. The patient was discharged on 10 Oct. with the grafted area well supported by crêpe bandages. After eight weeks the graft appeared healthy and showed only faint cyanosis (Fig. II).

FIG. II. Eight weeks after initial grafting, grafts appear to have taken well. There is a small marginal area of cyanosis surrounding grafts.

However, eleven weeks after this grafting one third of the grafted area sloughed, and two weeks later there was complete destruction of the remaining grafts. Coincident with this sloughing there was some oedema of the affected leg and ankle. After fifteen weeks the ulcer was larger than the initial lesion (20 x 15 cm.) but otherwise similar in appearance (Fig. III). Cortisone (75 mg. daily by mouth) was begun and three weeks later, on 3 Jan., split skin grafts from the left thigh were applied as previously to the base of the ulcer. All grafts survived and the total area was covered by epithelium in two weeks. The patient was allowed to walk and the grafts slowly improved in appearance (Fig. IV). During the ensuing six months the grafts remained intact, the oral administration of cortisone, 75 mg. daily, being maintained. He then died suddenly from coronary occlusion.

The necropsy showed changes of rheumatoid arthritis in the joints, and rheumatoid nodules. Histological examination of other organs revealed no further evidence of a lupus process. There was generalized atherosclerosis and recent occlusion of coronary vessels with myocardial infarction. The skin graft was completely intact and microscopic examination showed no cellular infiltration.

Summary of case. A 75-year-old man presented with chronic rheumatoid arthritis and recent ulceration of the leg which failed to heal with routine measures. The L.E. cell test was positive on two occasions and the AICF test was positive to a titre of 1 : 16. Split skin grafts to the ulcer sloughed after eleven weeks. The grafting was then repeated during continuous cortisone administration and the grafts survived and remained healthy.

FIG. III. Thirteen weeks after initial grafting. There is complete rejection of the graft with the reformation of a large ulcer.

DISCUSSION

We are reporting this case because our findings have theoretical and practical implications of considerable importance. Firstly, it should be emphasized that circulatory, neurogenic and trophic factors were, as far as possible, excluded as causes of the chronic ulcer. If, as suggested by Allison and Bettley (1957), we consider the features of this syndrome (rheumatoid arthritis, chronic ulcer of the leg and positive L.E. cell test) as a variant of systemic lupus erythematosus—a condition of suspected auto-immune aetiology—then the leg ulceration in this patient may be regarded as an immunologically determined lesion. Allison and Bettley (1957) also hinted at this

in suggesting that "local auto-immune disturbances" could be responsible for the ulcers of the leg in their own cases.

Circulating auto-antibodies were demonstrable in this patient, as evidenced by the repeatedly positive L.E. cell test and AICF test, but it is unlikely that any type of circulating antibody would react with a specific area of skin to produce this type of ulcer. It seems more likely that both the perpetuation of the ulcer and the failure of the initial autograft were dependent on the development of a type of vicious circle at the ulcer site. It is characteristic of systemic lupus erythematosus that the commonly present antibodies are directed against all components especially nuclear ones which are not accessible in intact cells. If reaction between antigen and antibody or between an immunologically reactive cell and antigen results in the liberation of cell-damaging agents, we have the possibility of a vicious circle. When a small amount of antibody or a few reactive cells can react with traces of antigen released by minor trauma or infection at the site of the potential damage, then the damage to adjacent cells may liberate more of the antigen to stimulate the production of more cells and antibody. These are attracted to the local site, and the vicious circle continues. We visualize the action of cortisone as limiting or even breaking this vicious circle.

The subsequent survival of an autograft under the influence of systemic cortisone therapy favours an immunological pathogenesis both for the original ulcer and the non-survival of the initial autograft. Although experimental data (Fischel, 1953; Trethewie, 1958) on the effect of cortisone on antibody production and hypersensitivity reactions are somewhat conflicting, we may accept the opinion of Taliaferro (1957) that cortisone does suppress immune responsiveness. Therefore, in the present case, cortisone might have been expected to suppress antibody production at the ulcer site to a degree sufficient to allow prolonged survival of an autograft with ultimate healing of the ulcer. Indeed, this is what took place.

From the practical viewpoint, we should advise that L.E. cell tests be performed on patients with severe rheumatoid arthritis and leg ulceration. A suggested plan of management of such cases would be to perform a skin autograft under optimal conditions. If the graft fails to survive, a second graft should be undertaken with the patient on maintenance cortisone treatment. It may well be that further experience with this plan will indicate the wisdom of grafting with cortisone as the *initial* procedure.

FIG. IV. Second graft applied when patient receiving oral cortisone. This graft remained healthy with continuing cortisone administration. This illustration was made during the sixth month.

SUMMARY

1. A report is given of an elderly male with rheumatoid arthritis, chronic ulceration of the leg and positive L.E. cell test, as described by Allison and Bettley (1957). In addition the auto-immune complement fixation (AICF) test was positive.

2. Split skin autografts to the ulcer failed to survive. When repeated during continuing cortisone administration they survived.

3. This experience favours an immunological pathogenesis both for the original ulcer and for the non-survival of the initial autograft.

4. A plan is submitted for the investigation of cases with rheumatoid arthritis and leg ulceration including an L.E. cell test. Treatment of such cases is suggested based on the use of cortisone if an initial graft fails to survive.

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A PROBLEM IN EVOLUTION

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I AM glad to offer my tribute to Sir Hugh Devine. Our College owes much to its founders and especially to that little group in Melbourne, who by virtue of personality and *locus*, had so much to do with its infant stages and the later problems of adolescence. Over and above his eminence as a surgeon, Sir Hugh must always hold high rank in our annals and we do well to honour him in this issue.

A surgeon of his quality, ripe age and wisdom, must have many thoughts when he surveys the progress of his fledgling over the past 32 years. Much has been accomplished and many problems still remain. This will always be so as surgery progresses, and to play its part, a College of surgeons must move in sympathy with the spirit of the times: indeed, it should recognize and even anticipate, changing ideas, sift them and build the good into the College structure.

Quickened by two great wars and maybe as an offset to the loss of potential talent that war exacts, this memorable century has produced many remarkable developments; remarkable to seniors nearing the end of their professional tether but almost commonplace to students and young graduates who have known no other.

Segregation and specialization

Not least remarkable is that evolutionary development through which numerous specialties have been created by a whittling away of the parent body, so-called general surgery. What was once an imposing edifice now closely resembles those dismembered trunks of dissecting room memory and the whittling still goes on.

In America for example, there is acceptance at high levels of the limitation of general surgery to the upper abdomen, thyroid and breast with pickings in the neck and limbs. So the parent body has become a specialty in itself and dismemberment has reached its apotheosis.

This plaint on the part of the general surgeon is no new thing for half a century ago a septuagenarian professor of surgery of my student days used to comment wryly: "The abdominal surgeons are moving down and the gynaecologists are working up. Soon there will be only a small area about the umbilicus left for the general surgeon." The hoary definition of a specialist as a man who knows more and more about less and less, expresses the same idea with trans-Pacific succinctness.

Be it as it may, specialization and segregation have come to stay in some form and have paid dividends through the more intense thought and the closer tilling given to restricted fields.

A parallel may be found in our two countries, certainly in New Zealand, in the breaking up of the great landed estates into smaller holdings, to the great benefit of production and a changed face to the countryside.

Resultant problems

But surgical segregation has brought to light new problems in its train. I shall limit my discussion to two only:

(a) First, in the active practice of surgery where too rigid an application brings many anomalies, absurdities and corners to be rounded off by time and good sense.

(b) Second, in the teaching of students and especially of aspirants to surgery, the surgeons of the future.

(a) The day-to-day practice of surgery

There is much to commend Harvey Cushing's concept that in the less explored and

more complex fields, surgery should be segregated and pioneered to put it on a sound basis; thereafter, what is appropriate should be passed back to general surgery, enriching it, not emasculating it. However sound and attractive, this idea does not work out in practice and is least applicable in Cushing's own field of neurosurgery. For many years the dividing line of sex has introduced an imaginary boundary between the compartments of the abdomen. The illogicality of the doctrine of the "indivisibility" of obstetrics and gynaecology needs no labouring but its acceptance tells us that what has happened in one field is likely to happen in another.

It is natural that the ebullience of a growing speciality should exploit possibilities at the outset but this urge for expansion should be guided if we are not going to dive off along spuriously attractive tangents. Let us consider some simple examples.

In thoracic surgery the present cult, which is likely to be strengthened in the minds of students and young graduates by the segregation they see practised in hospitals, that thoracic surgery *must* include all conditions in and about the chest, leads inevitably to this culmination; a goitre if it chances to lie within the thoracic cage, becomes, as it were, the property of the thoracic specialist. The good sense of it surely is that the general surgeon, accustomed to the frequent handling of goitre, is the right man to deal with it. Similarly with the thymus whose surgery has been put by Keynes, a general surgeon, on a sound basis; a kind of lend-lease gone wrong. The alimentary tube is peculiarly vulnerable to these segmental attacks because it chances to pass through chest, abdomen and pelvis. The day may come when thoracic surgery is restricted to the heart and great vessels, to the lungs and to such anomalies as congenital oesophageal fistula. The resources of modern anaesthesia, so important in this field, are available to all surgeons.

In orthopaedic surgery there has been a phenomenal growth and a spreading of tentacles till the original meaning of the name has been stretched to bursting point. It would seem reasonable to suggest a stock taking to decide what really demands specialist skill and what can be returned to general surgery. The fact is that each principality, like appetite, grows by what it feeds on and it extends its territories just as full-time professors are driven by an irresistible urge to spatial development. All surgery, as Brock insists in his special realm, is based on the same general principles; what we have to guard against (in my view) is the development of the one-eyed specialist to the ultimate detriment of surgery and a waning of its intellectual interest.

It remains to be seen whether the urge for segregation proves to be a passing phase which will run its course and, having played its part, will be followed by a secondary rise of an enriched general surgery with the cultivation of special "lines" of individual interest. The contributions of Moynihan and Walton in abdominal surgery, and Macewen and Ballence in neurosurgery, are examples in point from the past.

(b) The teaching of students and especially aspirants to surgery

In the undergraduate world the title "Professor of Surgery" has become something of a misnomer or will be as soon as the young, recently trained professors hold the field exclusively. "Director or Co-ordinator of Surgical Studies" is nearer the mark for his teaching mission is to inculcate sound principles, to expound such subject or subjects on which he can speak with authority and to dovetail the rest. In other words, he is no longer a one man textbook, so to speak, but has the hard job of editing his contributing specialists into a digestible and balanced whole for student pabulum.

The apprentice system in broad application has long been the keystone of British surgical education at all levels. Under the ruling wave of segregation, students in the "firms" of general surgeons are apt to have a sustained diet of gall-bladders, stomachs and colonic disorders, with a smaller helping of thyroid and breasts. The student now has to serve many masters with a consequent weakening of the master-apprentice link. But perhaps I make too much of it.

In the post-graduate field the College can exert a powerful directing influence in fashioning the new generation of surgeons in pursuance of its primary purpose to further

surgical education. One continues to hope that our purpose is to guide surgical aspirants up a graduated path in their own countries to a high level of competence; a programme which includes as a by-product so to speak, the acquisition of the diploma of F.R.A.C.S. This is no easy path for those who have the task of trying to fit the examinations, Primary and Final, and the necessary effective preparatory instruction for them, into an attractive and workable mosaic.

But let us leave matters of high policy and return more strictly to our theme.

If our purpose is to evolve a full surgeon in the Baconian sense, then the dangers of segregated surgery come into the picture, acting as it does upon the plastic mind of youth. There are two corrective avenues, at least.

(1) In the Registrar period (call it by what name is locally favoured), it becomes urgently necessary for the College, acting through its Fellows who control teaching in all branches of surgery in the larger centres, to have sufficient powers of direction to ensure that the surgical aspirant *rotates* through other services, whatever may be his chosen field. He will naturally spend most of his pupilage in this field, becoming more and more useful in the team and getting more and more to do; but at some stage, machinery must exist which allows him to be seconded for attachment to other units.

A year (at least) is not too much to allot for this purpose, in say, six month periods, over the whole training period. A smattering? Perhaps, but a useful insight if he practises in a metropolitan centre and invaluable if he goes to a counrty area, large or small. I grant its imperfections but in our countries we must try to make suitable provision for the many men who render invaluable service to the smaller communities. Both the surgeon and his community benefit greatly through this wider training, and the metropolitan surgeon is all the better for it.

I know there are difficulties in the application of this principle of rotation. No surgeon lkes to have his smoothly working team deranged but surgeons in large centres have a duty to further the broad training of our future surgeons and should accept inconvenience to this end. Trainees too, may be reluctant to follow any deviation from their chosen field. Consequently, it should be explained and accepted at the outset of their training programme.

(2) Those meetings which we slavishly call Staff Rounds (What is wrong with Surgical Staff Conference?), can play a useful, if minor part. Material should be deliberately selected, as far as it offers in clinical work, to cover the range of surgical practice for the benefit of the registrar group. For an hour a week he can then catch a glimpse of surgery as a whole and over the years this corrective dose surely must exert a useful wider educative influence.

To conclude this cursory discussion simply invites attention to a complex problem which lends itself to the nimble dialectics of debate and so we must leave it to the ruling wisdom of the day.

Shorn of verbiage, it may (if read) be rated, according to taste, as a cautionary or a reactionary tale.

Books Reviewed

THE PHYSICAL TREATMENT OF VARICOSE ULCERS.

By R. ROWDEN FOOTE. Edinburgh and London: E. & S. Livingstone, 1958. 7½" x 5", xii plus 125 pp., 88 figures. Price: 15s. (stg.).

This book is essentially a practical manual written for physiotherapists and nurses in order to give advice and assistance in the management of varicose ulcers.

Being written for physiotherapists and nurses, it does not deal with the surgical management of these lesions but lays down the principles which should be adhered to in the conservative management and the healing of ulcers before surgery is undertaken in selected cases.

The economic, anatomical and physiological considerations are discussed in the first three chapters. The division of hypostatic ulcers into "true varicose ulcers," resulting from incompetent superficial veins and "venous ulcers (post-phlebitic)," associated with the ankle blow out syndrome described by Cockett and Jones (1953) is the basis of future management. The former responding to ligation and stripping of superficial veins, the latter requiring ligation of perforating veins.

The principles of management indicate the importance of the control of oedema, which may be carried out by compression, at the same time increasing the activities of the patient, to prevent venous stagnation and to assist the calf muscle pump. The necessity to stimulate the patient's interest and optimism is not overlooked. All surgeons have seen these patients' fatalistic acceptance of their disabilities and apparent reluctance to improve the condition of their limbs.

Active treatment is discussed in detail, in particular the methods of compression. Whether this is carried out using dry (one way stretch) bandages, or moist (Elastoplast) bandages the principles of compression are identical. A very important practical adjunct is the use of pressure pads and of stockinette in eczematous or sensitized limbs to prevent further irritation from the bandages or elastoplast.

The technique of massage and physical exercises shown are carried out to reduce the oedema and to improve the activity of the patient. Overcoming the equinus deformity and ankle stiffness, so frequently seen in these patients, immediately assists the function of the calf muscle pump, reduces the oedema, relieves the pain and permits greater activity.

In the aftercare of healed ulcers the diagram showing the patient with knees fully extended between two chairs has perhaps been inaptly chosen. Phlebograms carried out in this position often show marked compression of the popliteal vein by the femur and thromboses have been attributed to patients resting in this position. It is better to have an elevated couch, supporting the whole leg and without compression behind the knees or calves.

This is an excellent book, well illustrated and outlining basic principles. It has been written for physiotherapists and nurses but practitioners who are in the habit of putting ulcer patients to bed, allowing the blood to stagnate and causing economic loss to the patient and community, would be well advised to study the book and to carry out the more active measures described. Rarely should it be necessary for a patient with a gravitational ulcer to require such bed rest.

EYE SURGERY.

By H. B. STALLARD, M.B.E., M.A., M.D., F.R.C.S., Hon.LL.D. Third Edition. Bristol: John Wright & Sons, 1958. 8½" x 5½", 916 pp., 671 illustrations. Price: 95s.

This book, which was first published in 1946, is now in its third edition and has become a standard textbook on operative ophthalmic surgery. It is particularly valuable in that it represents the personal experiences of a surgeon of Mr. Stallard's calibre. In addition, alternative operative procedures are described with comments, so that the reader is given a clear direction as to the most suitable operation for a given condition.

The book has been considerably enlarged. Much new material has been added on anaesthesia, particularly on general anaesthesia, which the author appears to favour. The subject of corneal grafting has also been considered in more detail than in previous editions, but the reader is left in doubt as to the author's own technique. For cataract extraction the author favours two preplaced corneal sutures and a Graefe knife section. The insertion of Ridley's acrylic lenticulus and the technique for the introduction of an anterior chamber lens are discussed in detail. The chapter on the surgery of the extra-ocular muscles is comprehensive, and the influence of Lyle and Chavasse is apparent. For squints having an onset at birth the author advocates operation at the age of one year.

The principles of plastic surgery, in so far as they concern the ophthalmic surgeons, are clearly shown, and much detailed description of war wounds present in previous editions has been omitted.

The book is a wonder of meticulous detail. Michaelangelo's words, "Success depends on detail," have evidently been the author's motto. This book is essential for every ophthalmic surgeon.

COMPARATIVE ANATOMY AND PHYSIOLOGY OF THE NOSE AND PARANASAL SINUSES.

By Sir VICTOR NEGUS. Edinburgh and London: E. & S. Livingstone Ltd., 1958. 10" x 7", xv plus 402 pp., 174 figures. Price: 70s. net (stg.).

We have always considered that the most original contributions to the teaching of practical anatomy in this century have been J. E. Fraser's *Anatomy of the Human Skeleton* (1914), F. Wood Jones' *Principles of Anatomy as Seen in the Hand* (1920), and Victor Negus' *Mechanism of the Larynx* (1929). Each of these authors was a man with a message and the presentation of his subject was novel and fascinating.

The present book may be regarded as complementary to the author's previous work on the larynx and follows the same lines.

The amount of research is amazing when one reflects that the author was busy and successful in private and public hospital practice and that the essential labour was done entirely by himself.

The book is divided into three parts, with an appendix, glossary and references.

Part I, entitled "The Comparative Anatomy and Physiology of the Nose," is subdivided into the following sections — the nose as an olfactory organ, the functions of the nose in respiration, the air conditioning mechanism of the nose, other functions of the nose, ciliary action, and exchange of fluid in the nose and respiratory tract.

Part II describes the comparative anatomy and physiology of the paranasal sinuses.

Part III describes the anatomy of the human nose and accessory sinuses.

The appendix discusses the evolutionary history of man from the evidence of the nose and larynx.

It is impossible to praise this book too highly or to exaggerate its importance as a contribution to our knowledge of the subject. We recommend it without reservation to appropriate readers.

There are 178 excellent illustrations of which only 26 have been borrowed or adapted from other authors.

The paper, binding and set up of the book are faultless, and are a credit to the publishers.

FRACTURES AND OTHER INJURIES.

By GEORGE PERKINS, M.C., M.Ch., F.R.C.S. London: The Athlone Press, University of London, 1958. 9¼" x 6", viii plus 363 pp., 255 figures. Price: Not stated.

The special feature of this unusual book is the advocacy of a minimum of rigid external splinting in certain selected fractures and early immobilization of the limb, free joints and muscles by active movements, especially if internal fixation is used. Every fracture is starred. One-star fractures can be treated by the patient's doctor, two-starred by a surgeon with facilities and three-starred fractures should be "off loaded" to the orthopaedic surgeon "because their treatment is difficult or because they have a dismal prognosis however well treated." Whilst much of the recommended treatment is in the orthodox mould, some is distinctly unorthodox. As these methods have caused a good deal of interest and have disclosed another facet of fracture treatment, some examples may be considered. In those compression fractures of the thoracic and lumbar bodies that he calls "stable" (no clinical gap in the interspinous ligaments and no radiographic "shift") the fracture should be disregarded and the patient taught to touch the toes by ten days. "Directly he can do this he needs no further treatment." He acknowledges "the treatment advocated above is not orthodox" but many good reasons are advanced to justify this course. If the fracture is "unstable" (interspinous ligament is torn), the advice is "stabilize in the routine way by bolting together the spinous processes." It is not stated whether any brace is used — presumably not. A shaft fracture of the humerus is immobilized simply by a sling. After three weeks the arm is put through a coat sleeve and the patient advised "to use it for everything." "While the patient is using the elbow he will hear crepitus in the first week or two, and it adds to his confidence to be warned of this by his doctor." "If union is slow, be patient, continue the sling for a longer period . . ." If plated, the shoulder and elbow are moved freely from the start. Strangely enough supracondylar fractures are immobilized by a posterior plaster slab "for greater security." Displacements are ignored in "T" shaped fractures of the condyles and congruity of articular surfaces is restored by early movements as operation too often results in a stiff joint. This is generally agreed. In the Monteggia fracture-dislocation prevention of redislocation of the head of the radius is claimed by flexion of the elbow, in supination, with a collar and cuff sling. This is the same as is recommended for a fracture of the shaft of the ulna about which the author provides this comment, "one is surprised that such a trivial fracture should take so long to repair."

In the lower extremity the same "delightfully simple" methods are used. For fractures of the femoral shaft the limb lies on a pillow placed lengthwise under the leg with skeletal traction for six weeks. The illustrations show considerable liberty, not only as regards knee movements, but also to the patient although skeletal traction is always maintained. In fractures of the tibial or femoral condyles splintage is by skeletal traction and the pillow for six weeks. The traction reduces the deformity, prevents valgus angulation and maintains the reduction. Knee movements are well established by ten days. It is generally agreed that by this method the functional results surpass those obtained by plaster immobilization or by operation. After nailing an intra-capsular fracture (two starred) the author admits, that, in theory, weight should not be taken under six months but adds "the patients who do well weight-bear on their own accord long before this." Failure may be caused by "the nail smashing its way out of the head" and this "is due to faulty technique and the surgeon is to blame." It is to be hoped that this statement does not become widely permeated in legal circles. A protest must be recorded that this view is not altogether true or widely held. Although it has to be admitted that if the nail is placed in the anterior half of the head it is likely to cut out through the bone in the anterior section of the caput femoris. A compound transverse fracture of the tibia and fibula (three starred) is treated in an orthodox manner. Provisionally, the leg rests on the pillow splint with skeletal traction of 10 lbs. And no definitive treatment is performed until the skin has healed and the reaction of the soft tissues to trauma has subsided. Then either a plate or plaster cast is applied from the middle of the thigh. After twelve weeks in plaster if there is callus a plaster gaiter from below the knee to above the ankle is applied. But if the fracture has been plated, a short plaster is applied from below the knee to the sole and early walking is urged after the stitches are out. In following the author's methods we now come to the ultimate in the ankle. Patients without subluxation of the talus "are left to look after themselves and they are got up without any splint." But, admittedly, a radiograph is advised in a week's time to check that no subluxation of the talus has taken place. When the talus is subluxated, either plaster fixation or, alternatively, internal fixation with long screws is recommended. "When the stitches are out, the patient is got up without external support and taught to walk." However, the subluxated ankle with vertical "T" shaped fractures of the tibia (three starred) is treated in skeletal traction and

no weight bearing for three months because the lower end of the tibia is so shattered.

This is an important book for those who wish to make a critical and fair study of the author's unorthodox work. The methods deserve consideration and good will come out of them as is shown in the management of condylar fractures of the femur or elbow and possibly the spine. But in other fractures such as the humerus or forearm and ankle joint, the conventional methods of splintage seem safer in the hands of most surgeons. Incidentally, there are only two three-starred fractures of the lower and one (scaphoid) in the upper limb.

A TEXTBOOK OF X-RAY DIAGNOSIS.

Volume 1—Head and Neck.
Volume 3—The Abdomen.

Edited by S. COCHRANE SHANKS and PETER KERLEY. Third Edition. London: H. K. Lewis & Co. Ltd., 1957. 10¼" x 7¾". Volume 1, xii plus 521 pp., 553 illustrations; Volume 3, xvi plus 883 pp., 802 illustrations. Prices: Volume 1, £4 10s. (stg.); Volume 3, £6 (stg.).

These volumes are new editions of the work so well known to all radiologists. The overall size of the books have been increased, giving wider margins and a more spacious layout. The index has been improved and the bibliography enlarged and brought up to date.

New contributors are Mr. D. W. C. Northfield, who has replaced the late Dr. Hugh Cairns, Dr. C. J. Hudson, Dr. D. Sutton, Dr. J. D. Drew and Dr. P. C. Keats.

Volume 1.—Part 1 is devoted to the central nervous system. The section on the radiological features of the cranium, normal and abnormal, is very little changed—any alteration is unnecessary as it is particularly well done.

Welcome additions are inclusion of the description of the anatomy of the intervertebral disc, an expansion of the section on intervertebral disc protrusion, and a new section on spondylosis.

Modern radiological procedure requires an enlarged description of the use of various opaque media in investigation of the central nervous system. The chapter on cerebral angiography is considerably expanded, both from the technical and diagnostic angles and is very fully illustrated. Myelography is also more fully covered. The use of the positive contrast medium "myodil" in ventriculography is described and the use of thorotrast pyography to demonstrate the extent and progress of cerebral and subdural abscesses has been included.

There has been practically no alteration to Part 2, devoted to teeth and jaws, and Part 3 on the eye. The illustrations in the former are of high quality.

Additional illustrations have been added to Part 4 on accessory nasal sinuses. The radiographic technique of sinus investigation is rather overdone and the section on the use of iodised oil in the investigation could now well be omitted being of little present practical interest.

Part 5 on the ear and temporal bone has some new reproductions of films of carcinoma of the middle ear, but is little altered otherwise.

Volume 3.—This volume includes the alimentary and biliary tracts the urinary system and radiological gynaecology and obstetrics.

There has been some expansion of the chapter on the oesophagus, neuro-muscular disorders and oesophagitis being more fully described in accordance with modern views.

The investigation of the stomach is well done. The difficulty of diagnosing some cases of carcinoma is stressed. The section on gastritis could well be omitted, the diagnosis being one for the gastroscopist rather than the radiologist.

In the chapter on the duodenum, post-bulbar duodenal ulcer is only mentioned to point out its rarity, but a little more emphasis on the necessity and methods of demonstrating it would be appreciated.

The section on hiatus hernia would be improved by emphasizing the difficulty in producing the hernia and a description of the ways in which it is most likely to be shown.

The chapter on the gall-bladder has been improved by the introduction of a description of the intravenous choledochographic agent and its use in the post-cholecystectomy syndrome. Mention is also made of the advantages of operative and post-operative cholangiography through the T-tube.

A section on portal venography has been introduced and the methods fully described. It is very well illustrated with some excellent reproductions.

The section on obstetrics and gynaecology is little altered. The use of improved media for hysterosalpingography is described.

The section on the urinary tract covers this subject adequately. The technique of investigation is very fully described, including the use of new drugs and an improved method of ureteric compression for use in intravenous pyelography, an essential factor in obtaining a satisfactory result.

The greatest criticism is the continued reproduction of positive films throughout both volumes—except in the part on teeth and jaws and some of the more recent illustrations. It would surely be worthwhile reproducing the negative radiographs to which everyone is accustomed.

The preface states that these volumes were conceived as a textbook to describe fundamental principles in interpretation and relate them to day-by-day clinical problems of the average X-ray department. This object is very well attained and these volumes remain the standard work for students of diagnostic radiology and are invaluable as reference books.

EMERGENCY SURGERY.

By HAMILTON BAILEY. Seventh Edition. Bristol, Eng.: John Wright & Sons Ltd., 1958. 10" x 6¼", xvi plus 1,197 pp., 1,576 illustrations, a number in colour. Price: £9 9s. (stg.).

The seventh edition of this familiar book has been extensively revised and the subject matter is now in one large volume. As before it is beautifully produced and illustrated and covers all conceivable surgical emergencies. The indexing is such that ready access to any problem is easily attained and since, with but few exceptions, one method only of performing any urgent operation is described, this book will be, as always, of great value to the junior surgeon and to any doctor called upon to treat surgical emergencies away from established centres of medical care.

One can have nothing but praise for this book which has enjoyed unrivalled popularity since it first appeared in 1930. It should be in the possession of every surgeon who is called upon to treat surgical emergencies.

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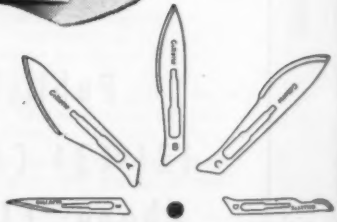

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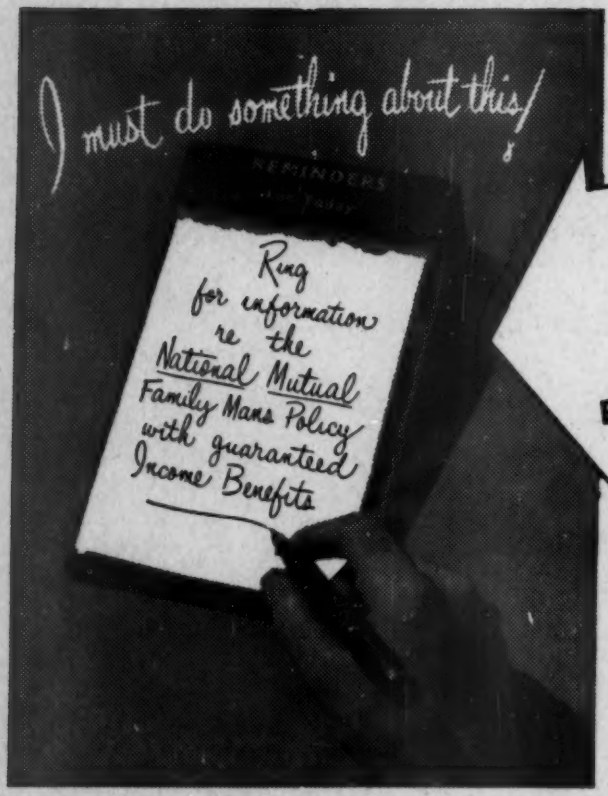
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